



This is a digital copy of a book that was preserved for generations on library shelves before it was carefully scanned by Google as part of a project to make the world's books discoverable online.

It has survived long enough for the copyright to expire and the book to enter the public domain. A public domain book is one that was never subject to copyright or whose legal copyright term has expired. Whether a book is in the public domain may vary country to country. Public domain books are our gateways to the past, representing a wealth of history, culture and knowledge that's often difficult to discover.

Marks, notations and other marginalia present in the original volume will appear in this file - a reminder of this book's long journey from the publisher to a library and finally to you.

### Usage guidelines

Google is proud to partner with libraries to digitize public domain materials and make them widely accessible. Public domain books belong to the public and we are merely their custodians. Nevertheless, this work is expensive, so in order to keep providing this resource, we have taken steps to prevent abuse by commercial parties, including placing technical restrictions on automated querying.

We also ask that you:

- + *Make non-commercial use of the files* We designed Google Book Search for use by individuals, and we request that you use these files for personal, non-commercial purposes.
- + *Refrain from automated querying* Do not send automated queries of any sort to Google's system: If you are conducting research on machine translation, optical character recognition or other areas where access to a large amount of text is helpful, please contact us. We encourage the use of public domain materials for these purposes and may be able to help.
- + *Maintain attribution* The Google "watermark" you see on each file is essential for informing people about this project and helping them find additional materials through Google Book Search. Please do not remove it.
- + *Keep it legal* Whatever your use, remember that you are responsible for ensuring that what you are doing is legal. Do not assume that just because we believe a book is in the public domain for users in the United States, that the work is also in the public domain for users in other countries. Whether a book is still in copyright varies from country to country, and we can't offer guidance on whether any specific use of any specific book is allowed. Please do not assume that a book's appearance in Google Book Search means it can be used in any manner anywhere in the world. Copyright infringement liability can be quite severe.

### About Google Book Search

Google's mission is to organize the world's information and to make it universally accessible and useful. Google Book Search helps readers discover the world's books while helping authors and publishers reach new audiences. You can search through the full text of this book on the web at <http://books.google.com/>

LANE MEDICAL LIBRARY STANFORD  
P45 .X63 1902  
The diseases of infancy and childhood :



24503435063







**THE DISEASES**  
**OF**  
**INFANCY AND CHILDHOOD.**

**DESIGNED FOR THE USE OF**  
**STUDENTS AND PRACTITIONERS OF MEDICINE.**

**BY**  
**HENRY KOPLIK, M. D., 1855-**

**ATTENDING PHYSICIAN TO THE MOUNT SINAI HOSPITAL; FORMERLY ATTENDING PHYSICIAN  
TO THE GOOD SAMARITAN DISPENSARY, NEW YORK; EX-PRESIDENT OF THE AMERICAN  
PEDIATRIC SOCIETY; MEMBER OF THE ASSOCIATION OF AMERICAN PHYSI-  
CIANS, AND OF THE NEW YORK ACADEMY OF MEDICINE.**

**ILLUSTRATED WITH 169 ENGRAVINGS AND 30 PLATES IN  
COLOR AND MONOCHROME.**



**LEA BROTHERS & CO.,**  
**NEW YORK AND PHILADELPHIA.**

---

Entered according to Act of Congress, in the year 1902, by  
LEA BROTHERS & CO.,  
In the Office of the Librarian of Congress. All rights reserved.

---

---

WESTCOTT & THOMSON,  
ELECTROTYPERS, PHILADA.

---

WILLIAM J. DORNAN,  
PRINTER, PHILADA.

#83

1-2-22

**Arthur Lee Clark**

**THIS WORK**

**IS INSCRIBED TO MY PRECEPTORS,**

**FRANCIS DELAFIELD, M.D., LL.D.,**

**EMERITUS PROFESSOR OF THE PRACTICE OF MEDICINE IN THE COLLEGE OF  
PHYSICIANS AND SURGEONS, COLUMBIA UNIVERSITY, NEW YORK,**

**AND**

**MATHEW D. MANN, M.D.,**

**PROFESSOR OF OBSTETRICS AND GYNÆCOLOGY IN THE UNIVERSITY  
OF BUFFALO, NEW YORK.**



## PREFACE.

---

DURING the past decade scientific research in medicine has been especially active in the domain of Pediatrics. The literature of the subject has grown luxuriantly on both sides of the Atlantic. Much of it exists in monographs and special papers, and is thus scattered and inaccessible by those conversant with the English language alone. The time, therefore, seems opportune for a work which should endeavor to gather and unify the world's best practice in a systematic and convenient volume.

In the following pages, accordingly, American, English, French, German and Italian pediatric science is fully represented. The work is, however, not in any sense a compilation. It is based upon the author's individual experience and his careful judgment regarding the work of other pediatricists. He has endeavored to spare his readers the labor of deciding between divergent views, and has adhered to his purpose of affording the physicians and students of his own country a practical guide and text-book.

He desires to express his gratitude to his publishers for many valuable suggestions.

H. K.

NEW YORK, August, 1902.





# CONTENTS.

## CHAPTER I.

### INFANCY AND CHILDHOOD.

	PAGE
Definition of infancy and childhood—Morbidity—Mortality—Methods of examination: taking a history—The head—The face—Sight—Physical examination of the chest: position of the patient; instruments used; methods of procedure: palpation; percussion—The abdomen: inspection; rectal exploration—Examination of the joints—The spine: anatomy; methods of examination—Muscular apparatus and nervous system: form; atrophy; hypertrophy; muscle-reflexes; Babinski's reflex; Kernig's symptom; gait and walk— <i>Administration of drugs and other methods of therapy</i> —Hydrotherapy—Hypodermoclysis—Syringing the nose—Vapor spray and calomel inhalations in acute laryngeal disease—Stomach-washing—Gavage—Rectal enemata, irrigation, enteroclysis—Hygiene of infancy and childhood: the first bath; the eyes; the daily bath; the temperature of the room; the body binder; clothing; the skin; the mouth; the diaper; the temperature; the breasts of the newborn infant; open air; sight; hearing; standing and walking— <i>Natural feeding of infants and children</i> —Selection of a wet-nurse: quantity of milk—The milk is insufficient in quantity—Contraindications to nursing—Placing the baby at the breast—The care of the breast: caking; sore nipples—Nursing the infant: intervals of nursing; signs of efficient nursing; increase of weight; movements of the bowel; signs of insufficient breast-feeding—Composition of human milk: alteration of the constituents of mother's milk; influence of food on breast milk—Bacteria in breast milk—Methods of analysis of human milk: specific gravity; fat; proteids—Microscopical examination of human milk— <i>Artificial feeding of infants</i> —Composition of cows' milk; the increase in weight of artificially fed infants—The modification of cows' milk for infant feeding: the whey method of dilution and modification—Bacteria in cows' milk: pasteurization; sterilization—Raw milk in infant feeding—The nursing bottle—Quantity to be fed to the infant—Amount of breast milk consumed by the infant in twenty-four hours—Number of nursings daily in artificially fed infants—Composition of the food—Household modification of milk for infant feeding—Infant foods—Dextrinized gruels as an infant food—Other foods: barley-water; albumin-water; peptonized milk; koumyss; beef-juice; acorn cocoa—Feeding of breast-fed infants and of bottle-fed infants after the sixth month—Feeding from the ninth to the twelfth month—Feeding from the twelfth to the eighteenth month—Feeding from the eighteenth month to the end of the second year—Feeding of sick infants and children . . . . .	17-84

## CHAPTER II.

PREMATURE INFANTS—DISEASES OF THE NEWBORN INFANT—  
INJURIES INFLICTED DURING BIRTH.

Premature infants: management of premature infants; incubators—*Asphyxia* of the newborn infant—*Asphyxia* subsequent to birth—*Atelectasis* of the lung—Septic infection of the newborn infant—Diseases of the umbilicus—Umbilical Fungus—Infection of the umbilical vessels—Hemorrhage from the umbilicus—Idiopathic hemorrhage from the umbilicus—Umbilical herniæ—Melæna neonatorum—Acute fatty degeneration of the newborn infant—Winckel's disease—Tetanus of the newborn infant—Ophthalmia neonatorum—Icterus in the newborn infant—Sclerema—Injuries inflicted during birth—Hæmatoma of the sternomastoid muscle—Cephalohæmatoma . . . . . 85-1

## CHAPTER III.

## THE SPECIFIC INFECTIOUS DISEASES.

The Exanthemata—Scarlet fever—Rötheln—Measles—Varicella—Vaccination—*Other specific infectious diseases*—Typhoid fever—Malarial fever—Influenza—Glandular fever—Cerebrospinal meningitis—Posterior basic meningitis—Mumps—Pertussis convulsiva—Diphtheria—Diphtheroid—Scrofula—Tuberculosis: Pulmonary tuberculosis—Tuberculosis of the peritoneum—Simple chronic peritonitis; other forms of tuberculosis (larynx; pleura; pericardium)—Abdominal tuberculosis—Tuberculous meningitis—Tuberculosis of the brain—Syphilis: Acquired syphilis—Late hereditary syphilis—Congenital or hereditary syphilis . . . . . 118-2

## CHAPTER IV.

## DISEASES OF THE MOUTH, PHARYNX, AND LARYNX.

Characteristics of the normal mouth—Dentition: rachitis; syphilis; Hutchinson's teeth; dental erosions; microdontism; dental infantilism; amorphism—Pathology of dentition—Aphthous stomatitis—Bednar's aphthæ—Sprue—Toxic stomatitis—Ulcerative stomatitis—Pseudodiphtheritic stomatitis—Gonorrhœal infection of the mouth—Noma—*The tongue*—Congenital anomalies of size (macroglossia)—Ringworm of the tongue—Desquamation of the epithelium of the tongue—Tongue-swallowing—Tongue-tie—*Malformations of the urula*—Acute retropharyngeal abscess—Retro-œsophageal abscess—Adenoid vegetations—*The tonsils*—Acute follicular amygdalitis—Herpes of the tonsils—*The larynx*—Acute catarrhal laryngitis . . . . 278-30

## CHAPTER V.

## DISEASES OF THE GASTRO-ENTERIC TRACT.

Physiological and anatomical facts concerning digestion in infants and children—The stomach: capacity; function and motility; reaction; marking out

	PAGE
the stomach by percussion—Intestinal ferments—The liver; the gall bladder; the pancreas; the bacteria—Characteristics of the stools of infants—Acute gastric dyspepsia—Habitual vomiting of infants—Colic—Tympa- nites—Hypertrophic pyloric stenosis—Acute gastro-enteritis—Cholera in- fantum—Gastro-intestinal atrophy—Acute and subacute enterocolitis—Dys- entery: amœbic dysentery—Constipation and mucous disease or mucous colitis—Acute intestinal obstruction—Appendicitis: acute; chronic— <i>The rectum: anatomical</i> —Prolapsus ani—Fissure of the anus—Spasm of the anus—Proctitis—Polypus of the rectum—Intestinal parasites . . . .	305-356

## CHAPTER VI.

## DISEASES OF THE RESPIRATORY TRACT.

General considerations: movements of the chest; fremitus; normal limits of the lung; resiliency of the chest wall; pulmonary resonance; dullness; tympanitic resonance; auscultation; the types of breathing; forms of dyspnoea—Acute simple bronchitis—Fibrinous or plastic bronchitis—Em- physema and chronic bronchitis of the lung—Bronchiectasis, including putrid bronchitis—Lobar pneumonia—Bronchopneumonia—Persistent bron- chopneumonia—Pleurisy—Dry pleurisy—Pleurisy with effusion and empy- ema—Perforating empyema—Hemorrhagic pleurisy—Hemorrhagic empy- ema—Subphrenic abscess or pyopneumothorax subphrenicus . . . . 357-432

## CHAPTER VII.

## DISEASES OF THE HEART AND PERICARDIUM.

The heart: position; size; apex-beat; inspection; palpation; auscultation; percussion; locating the line of dullness of the left ventricle; the pulse; the rapidity; the rhythm—Congenital heart disease—Stenosis of the pul- monary artery, conus or ostium—Open ductus arteriosus or ductus Botalli— Congenital defects of the auricular ventricular septum, defects of the auri- cular septum, open foramen ovale—Congenital deficiency of the ventricular septum (*Maladie de Roger*)—Acute endocarditis—Septic, ulcerative, or malignant endocarditis—Chronic heart disease—Cardiac murmurs—Acci- dental cardiac murmurs in infancy and childhood—Myocarditis—Pericar- ditis—Adherent pericardium—Hypertrophy and dilatation . . . . 433-467

## CHAPTER VIII.

## DISEASES OF THE NERVOUS SYSTEM—METHODS OF DIAGNOSIS.

Lumbar puncture—Normal cerebrospinal fluid: abnormal conditions; the spe- cific gravity; the gross appearances; tuberculous meningitis; suppurative meningitis; epidemic and sporadic cerebrospinal meningitis; chronic hydro- cephalus; the sediment; the pressure—The operation of lumbar puncture —Infantile convulsions—Hysteria—Tetany—Catalepsy—Myotonia—Con-

genital stridor of infants—Laryngismus stridulus—Epilepsy—Pavor nocturnus—Chorea—Chorea insaniens—Forms of Tic—Rhythmic movements of the head associated with nystagmus—Hydrocephalus—Acute hydrocephalus—Chronic internal congenital hydrocephalus—External hydrocephalus—Amaurotic idiocy—Tumors of the brain—Infantile cerebral palsy—Facial palsy—Multiple neuritis—Erb's palsy—Hereditary ataxia—Acute atrophic paralysis—Juvenile form of progressive muscular atrophy—Landouzy's form of muscular atrophy—Pseudohypertrophic muscular paralysis—Deformities of the skull and spinal canal—Spina bifida . . . . .	PA 468-51
---	--------------

## CHAPTER IX.

### GENERAL DISEASES.

Rachitis—Rheumatoid arthritis—Acute articular rheumatism—Rheumatoid affections—Diabetes mellitus—Diabetes insipidus . . . . .	533-54
---	--------

## CHAPTER X.

### DISEASES OF THE LYMPH NODES, DUCTLESS GLANDS, AND DISEASES OF THE BLOOD.

The lymph nodes (infections of)—Acute adenitis—Chronic lymphadenitis— <i>Diseases of the thyroid gland</i> —Cretinism (endemic and sporadic)—Fœtal rickets or achondroplasia—The thymus gland: landmarks: weight: percussion—Hypertrophy of the thymus— <i>The spleen</i> : anatomical: percussion: palpation—Splenic and kidney tumors— <i>The blood</i> —Leading characteristics in infancy and childhood: the red blood-cells: the white blood-cells: the hæmoglobin: the specific gravity—Anæmia—Simple anæmia—Chlorosis—Pseudoleukæmic anæmia of von Jaksch—Leukæmia—Acute leukæmia—Chronic leukæmia—Status lymphaticus—Hemorrhagic diathesis—Simple purpura—Hæmophilia—Purpura hæmorrhagica—Purpura rheumatica—Henoch's purpura—Pernicious anæmia—Infantile scorbutus or scurvy . . . . .	556-56
---	--------

## CHAPTER XI.

### DISEASES OF THE BONES.

General facts concerning diseases of the bones—Osteomyelitis—Otitis in infancy and childhood—The mastoid region: general facts: mastoid disease . . . . .	595-60
---	--------

## CHAPTER XII.

### DISEASES OF THE LIVER.

Anatomical facts: method of examination—Jaundice—Cirrhosis of the liver—Abscess of the liver—Fatty degeneration of the liver—Syphilis of the liver—Acute yellow atrophy of the liver . . . . .	607-61
--	--------

CONTENTS.

xi

CHAPTER XIII.

DISEASES OF THE KIDNEYS.

	PAGE
Anatomical facts—The urine—Cyclic albuminuria—Edema or hydremia without kidney lesion—Dysuria—Hæmaturia—Hæmoglobinuria—Renal calculi—Acute nephritis—Chronic nephritis—New growths of the kidney—Cysts of the kidney—Hydronephrosis—Sarcoma of the kidney—Carcinoma of the kidney—Tuberculosis of the kidney—Pyelitis (pyelonephritis)—Perinephritis and paranephritis—Enuresis nocturna and diurna—Vulvovaginitis—Urethritis in male children—Cystitis . . . . .	614-638

CHAPTER XIV.

DISEASES OF THE SKIN.

General facts—Eczema—Erythema multiforme—Furunculosis—Sudamina—Dermatitis exfoliativa—Congenital ichthyosis—Pemphigus neonatorum . . . . .	639-650
--	---------



# DISEASES OF INFANCY AND CHILDHOOD.

## CHAPTER I.

### INFANCY AND CHILDHOOD.

#### DEFINITION OF INFANCY AND CHILDHOOD.

**Infancy**, or the nursing age, is the period of life during which the child is at the breast. It extends from birth to the twelfth month.

**Childhood** is the succeeding period, extending to the tenth year. In addition, it is customary to divide the period of childhood into two parts—the first extending from the end of the first to the fifth year; the second, from the fifth to the tenth year.

Epstein would include as **newborn** all infants up to the third month.

#### MORBIDITY.

**The Newborn Infant.**—The diseases of the newborn infant are, for the most part, septic in nature, and attack the infant within a short time after birth.

Conditions favor the diseases common at this time of life. The skin is not fully formed, is in process of desquamation, and bacteria obtain entrance. The umbilicus is an open wound, receptive of infection. The mucous membranes of the intestine, mouth, eye, and ear are other avenues of entrance for bacteria. There is a tendency for minor infections to become general at this period. The artificially fed infant is, in addition, exposed to the dangers which necessarily accompany the introduction into the body of a foreign food with its attendant uncleanness, and is also deprived of the protective bodies (antitoxins) contained in the mother's milk. With new surroundings, in a new atmosphere, with new appliances for maintaining the body-heat (such as the clothes), and with careless handling, it is obvious that the newborn infant is particularly subject to bacterial diseases.



**Childhood.**—If we study the statistics of any large pediatric clinic, it will at once be apparent that up to the tenth year of those diseases which affect the respiratory apparatus form nearly two-fifths of the cases. Next in order of frequency are the diseases of the digestive tract; and, lastly, the acute infectious diseases, such as the fevers and exanthemata. Of 53,040 cases met with during the first ten years in an ambulatory clinic, there were 20,207 cases of diseases of the respiratory organs, 17,058 of the gastro-enteric tract, and 15,775 of the acute infectious diseases. If the morbidity is analyzed still further, it is seen that in the nursing period intestinal disturbances are the most frequent. The numerous flora of bacteria and their toxins in the intestine of the infant rather predispose to infections from that source. These bacteria may invade the mucous membrane of the infant, and in certain disturbances of the function of the gut obtain access to the circulation. The respiratory diseases become more frequent in the second year, and reach their maximum between the second and third year. Constitutional diseases, such as rickets, appear in the second half-year of life, and reach their greatest frequency during the period from the tenth to the fifteenth month. On the other hand, the acute infectious diseases, such as the exanthemata, are more common from the fifth to the eighth year. Scarlatina, with its kidney complications, is most frequent at the fourth year (Escherich), diminishing at the ninth year. The period extending from the second to the fourth year is also notable for the frequency of the so-called "filth infections" of Feer. Children infect themselves with dirt and dust, at play, at meals, or in the intercourse with one another. For this reason, diphtheria as well as pertussis and tuberculosis (Escherich) attain their maximum frequency at this period.

### MORTALITY.

The mortality of infants is large, and reaches its highest figure among the poor of large cities. Among the wealthy, artificial feeding is resorted to for social reasons; among the poor, a mother who is forced to work is compelled to deny the breast to her child. The vast majority of deaths occur among artificially fed infants. In England fully two-fifths of the whole number of deaths occur before the tenth year, one-fourth occurring before the termination of the first year. These figures, given by Williams, correspond closely to those of Eröss in Germany, and to what is known to be true of America. The mode of living among the poor, and the lack of complete or of even partial isolation in infectious diseases, tend to increase this great mortality among them.



**METHODS OF EXAMINATION.**

**Taking a History.**—Beginning with a few leading questions, the physician inquires as to the sex and age of his patient, the number of children in the family, and the use of instruments, such as forceps, in the delivery of the infant. The methods of feeding the infant from the outset, are inquired into, and the success attending these methods. If the patient is in the period of dentition, the order of the eruption of the teeth is ascertained. After eliciting information in regard to any previous illness, the physician proceeds to the details of the existing affection. In the greatest majority of cases an illness in infants begins with fever, chill, cyanosis, or vomiting. One of these symptoms may be present to the exclusion of the others, or they may all be present, or the illness may be ushered in with a convulsion. The condition of the patient immediately following the initial symptom constitutes the initial stage of the illness. Fever or unconsciousness may follow a chill or convulsion, or the patient may after the initial symptom develop an eruption, cough, dyspnoea, or pain. The fever may subside in a few hours, and the temperature return to normal, with a subsequent rise, preceded by a chill, cyanosis, or a second convulsion. Older children may complain of pain, as adults do. In the case of an infant, pain in the chest or abdomen may be indicated by an increase in the number of respirations or a sighing or moaning with each effort at respiration.

The vomiting of the initial stage of the illness may not be repeated, or it may recur and form a leading feature. The nature of the vomited matter is important. It may have an acid reaction or odor, or may consist of stomach contents mingled with biliary pigment. It may be streaked with blood. In serious continued vomiting it may assume a fecal character. Vomiting may occur with the ingestion of food or independently of it.

The condition of the bowels is of importance. The movements may be numerous but of normal consistency and odor, or they may be diarrhoeal and have abnormal features. The movements may be accompanied by tenesmus or prolapse of the gut. The urine of sick infants is sometimes not passed for hours. The mother will make a note of this fact. The character of the urine is next to be ascertained. Its passage may be painful. The urine may stain the diaper yellow (jaundice) or red (lithiasis); it may contain blood. Older children may be required to pass the urine. The quantity is more easily estimated in older children than in infants. With the latter we should be cautious in drawing conclusions as to the daily amount. In taking a history as above, it is essential, while eliciting the main features of an illness, not to inquire concerning unimpor-

tant details. The main features of the history should be grasped and completed in all their minutiae.

**Taking the Status Præsens.**—It often happens that the infant or child is asleep during the first portion of the visit. Under this condition the respirations and pulse, with the character of each, should be noted. The posture during sleep, the expression of the face, the contour, the position and behavior of the extremities during wakefulness are of the greatest import. Respiration during rest is more insidious than in a condition of unrest and wakefulness. The patient should be completely undressed for examination. This is done as a routine procedure even in cases of apparently mild illness. A skin eruption on the skin is thus forced upon the attention of the physician.

### The Head.

The examination of the head should begin with observation of its size, whether normal or abnormally small or large. The general shape of the head and condition of the bones are of importance. Reference to the presence or absence of rachitis and areas of craniotabes. The manner in which the head is held is noted, bearing on the presence of torticollis. In Pott's disease the head is held rigidly on the spine, and in older children supported by the hands. Some infants, for instance, amaurotic idiots and those suffering from birth-paralyses or diphtheritic paralysis, are unable to hold the head upright. In forms of meningitis the head is retracted or held rigidly. The fontanelles may be normal, tense, depressed, or abnormally prominent; they may be closed prematurely or open beyond the normal period. The presence of tumors underneath the scalp should be noted. The condition of the lymphatic nodes posterior and anterior to the border of the sternomastoid muscle is of clinical importance.

### The Face.

The expression of the face in a condition of rest, and also when the infant or child cries, may enlighten us as to the presence or absence of paralyses. These may be localized, involving the muscles of one organ, such as the eye, or the whole side of the face may be affected. When the infant is asleep the mouth is normally closed and the infant breathes through the nose, the tongue being applied to the roof of the mouth. In abnormal states the breathing may be noisy; the cry may be peculiar, as described under Retropharyngeal Abscess; the lips may be cyanosed or the seat of rhagades or eruptions, such as herpes; the symmetry of the face may be lost, as in parotiditis or adenitis, in which there is a swelling of one or both sides of the face.



**Cardiac disease** in advanced stages gives a sad and anxious expression to the countenance.

**Facial paralysis**, either partial or complete, causes a characteristic facial expression. If the infant cries, or the child is made to smile, one side of the face remains immobile. Even in rest the angle of the mouth may be drawn toward the unaffected side of the face, as in tuberculous meningitis.

In **nuclear palsy** of the congenital variety described by Moebius and Schapring (pleuroplegia) both sides of the face are immobile, and the face has a mask-like expression. There are no folds in the face either in the acts of laughing or crying.

**Basedow's disease** gives a peculiar expression to the face, caused by the prominent eyeballs, which are pathognomonic of this disease.

**Hydrocephalus** likewise gives a peculiar facial expression. The forehead is protuberant and overhanging. The eyeballs are forced downward, and the sclera are seen. The face proper is small as compared to that part of the head above the eyes. This is due to the large size of the cranium.

**Rachitis** at times causes a characteristic expression which is likely to be confounded with that due to hydrocephalus. In some rachitic infants the eyes are prominent and the sclera can be seen slightly. The orbital plates of the frontal bone being thin, the weight of the brain depresses the eyeball to a very slight degree.\*

**Exhausting diseases**, such as diarrhoea, cause prominence of the eyes, giving a very characteristic expression—the so-called hydrocephaloid of older writers.

**Congenital syphilis** in some cases causes a deformity of the nose, which is present at birth. The result is a peculiar angular deformity of the normal nasal curve. Looked at sideways, the bony septum is depressed; the cartilaginous septum is still intact. A very acute angle between the two results. This is similar to what is seen in destructive forms of syphilis later in life. The facial expression is characteristic of the disease.

The angle of the **palpebral fissure** is altered in conditions such as Mongolian idiocy. In this affection it is slightly oblique. In paralyzes of the ocular muscles the palpebral fissure itself may be wider in one eye than in the other. The presence or absence of conjunctivitis, keratitis, nystagmus, paralyzes of the orbital muscles, the condition of the pupils, are all points of importance in determining the status presens. In diseases of the brain or its coverings an ophthalmoscopic examination of the fundus oculi should be made.

### Sight.

In partial or total blindness, not only do the patients not notice objects placed in front of them, but there is in addition a vacant

expression or stare. If the blindness is total, the finger will be suffered to approach the eye so as to touch the cornea.

Some infants have a tendency to hold the head to one side. This may be due to defective vision or to weakness or spasm of the muscles of the neck. In cases of defective vision the head assumes a normal position if the eyes are not focussed on any object. As soon, however, as an effort is made to see, the head is inclined so as to bring the planes of vision of the eyes in accord.

**Photophobia** is an aversion to light, and is due to a spasm of the ocular sphincter in diseases of the conjunctiva or cornea (conjunctivitis, corneal ulcer).

**Nystagmus** is a series of involuntary movements of the eyeball, due to inefficiency of certain muscles, and is met with in conditions of corneal opacity, congenital cataract, albinism, infantile amblyopia, spasms, nutation or head-nodding, and in nervous states, such as amaurotic idiocy. In weakly rachitic infants nystagmus may be exhibited around a horizontal or vertical axis of the eyeball, or it may show itself in a rotary oscillation of the globe. It is made manifest in infants by causing them to focus some bright object, held slightly above and to one side of the head.

### Physical Examination of the Chest.

**Position of the Patient.**—An infant should be so held for examination that the examiner and the patient may be at ease. Being undressed, with the thorax exposed, the infant is first held by the attendant with its head looking over her shoulder, in which position the arms instinctively clasp her neck (Fig. 1). The patient so placed does not see the examiner. The spine should be straight, so that in percussing the sound is obtained on both sides under the same conditions. To examine the chest anteriorly, the infant is held looking forward, the anterior aspect of the thorax facing the examiner. If it is able to sit up, it may be examined in the sitting posture, both anteriorly and posteriorly.

With older children it is best to make an examination with the patient sitting upon a table or chair in a position convenient to the examiner. If confined to bed, the child must be examined in bed. As a rule, however, it is preferable to have the patient taken out of bed into the light.

Infants and children sometimes try to grasp the instruments of the examiner; gentle suasion will reassure them, force is never necessary.

**Instruments Used.**—A **stethoscope** is absolutely essential to the proper examination of the chest of an infant or child. This method is called **mediate examination**. We can by its means assure ourselves that the whole area of the chest has been carefully investigated.



Examination by the ear—the immediate method—is uncertain. A small area of bronchopneumonia may easily escape detection in infants and children of tender age, in whom the axillæ and lateral regions of the chest should be carefully searched. Direct application of the ear to the chest is resented by infants and children, and is not a convenient procedure for the physician. With the stethoscope he can follow the movements of the body of a restless patient.

FIG. 1.



Method of holding the infant for the examination of the posterior portion of the chest and lungs.

The best form of stethoscope to employ is the binaural. The instrument devised by the author (Fig. 2) has given him the most uniform results. A larger stethoscope, such as that employed for examination of the adult chest, does not differentiate the variety of sounds as well as this small instrument, and may cause pain to a restless infant, inasmuch as the chest-piece must be held too rigidly and is likely to press painfully against the chest-wall. The old form of stethoscope, consisting of a rigid wooden or hard-rubber tube applied to the ear, should not be used. If the head is pressed too

forcibly against the instrument, the pressure is communicated to the chest and will make the infant unruly.

A **steel tape-measure**, marked off into inches and centimetres, is convenient for detecting inequalities in the size of the sides of the chest.

**Methods of Procedure.—Inspection.**—We learn by inspection the shape of the chest and the character of the respiratory movements; also, the aspect of the cardiac area, the pulsation of the apex of the heart, its force and situation.

Respiration in infants and children is of the abdominal type. The rapidity may be counted by noting the movements of the chest or by watching the rise and fall of the epigastric region in the recumbent patient.

**THE CARDIAC AREA.**—In some infants and children the cardiac area may be quite prominent without the presence of any cardiac disease. In rachitic infants and children this part of the chest-wall may conform to the shape of the heart. There remains even in the later childhood of rachitic patients a very slight rotundity or fullness

FIG. 2.

Author's form of stethoscope. (*Archives Ped.*, Nov., 1899.)

of the precordial region. If the chest-wall is quite thin, the precordial region may normally present a wave of pulsation. All these signs may be exaggerated in disease of the heart. The apex-beat is normally distinguishable. Its force and area may be increased or diminished in disease. The apex-beat may be displaced upward and outward, or inward toward the median line (conditions of effusion in pericardium or pleura).

**Palpation.**—Palpation, by laying the palmar surface of the hands on the chest, is hardly to be attempted with young infants and children. In these subjects the chest is so small that this method cannot mark out areas of fremitus or absence of the same. To determine its presence, it is more satisfactory to use the internal border of the hand, generally the right. The hand is held horizontally, the internal border pressing firmly against the chest-wall. Thus the slightest variations in vibration of the chest-wall can be detected. We begin above at the upper border of the chest and pass downward, comparing both sides. If the infant or child cries, so much the better. If we wish to ascertain the presence of fremitus



in a baby, we may even cause it to cry by a procedure which will be described in the chapter on Diseases of the Lungs and Pleura. Older children may be asked to count or induced to talk. In infants and children fremitus is not so marked or useful a sign as in the adult. Normally, it diminishes in intensity as the base of the lung is approached. In some children it is detected in the lower part of the thorax only by careful examination. It is normally well marked along the axillary line; it is most marked along the mid-regions of the chest between the scapulae behind. Anything which separates the lung from the chest-wall will diminish or extinguish fremitus. Solidification of lung tissue will cause better conduction and increase it.

**Percussion.**—It is not advantageous to use a pleximeter in examining infants and children. The index finger of the left hand is laid horizontally on the chest with firm pressure. The skin or chest-wall and finger are thus made one medium. Percussion is performed by making a hammer of the middle finger of the right hand. The force used should come from the wrist; the forearm should be immobile. The stroke is expended upon the middle phalanx of the finger on the chest-wall, and should be of a tapping character, similar to that used in striking the keys of a typewriter; there should not be a pushing motion. The force should not be great. A force equal to that necessary in the examination of the adult chest would set in vibration all the neighboring chest and abdominal organs and cavities, and would not bring out the delicate distinctions of sounds necessary to diagnosis. Moreover, to rachitic infants and young children a forcible stroke is distinctly painful.

### The Abdomen.

The abdomen of an infant or child is best examined with the patient on a bed or a table covered with a soft blanket. The mother's or nurse's knees are not so satisfactory a surface for this purpose. The patient should be completely undressed.

**Inspection** should include the examination of the skin as to color, eruption, presence or absence of œdema, and of the abdomen as abnormally rotund or relaxed. In the latter condition we may sometimes make out the coils of gut. In diseases which exhaust the strength of the patient we distinguish between relaxed and retracted abdominal walls. A retracted abdominal wall may be tense and incurvated—the so-called boat-shaped abdomen; this is seen in meningitis. In some rare forms of septic peritonitis also the abdomen may be retracted. The pain of a colicky attack will cause the abdominal walls to be tense although not retracted. In intussusception the coils of gut or even the intestinal tumor may be seen on the surface. Ascites distends the abdomen, and when marked the rotundity is characteristic, and the skin is tense and shining.

**Peritonitis** causes tympanitic distention. In perforation of gut in typhoid fever or appendicitis the tympanites is accompanied at an early stage, as in the adult, by disappearance of the liver dullness. This sign will aid us more if the liver dullness and flatness have been determined accurately in advance of any complication.

**Tumors.**—Abdominal tumors give an uneven contour to the abdomen. Such tumors are met in diseases of the spleen or kidney (sarcoma). Enlargements of the liver and spleen give similar appearances. Congenital renal cysts, ovarian tumors, or hydatid cysts cause uneven distention of the abdomen.

**Palpation.**—We palpate for pain, general or localized, and determine the size and position of the abdominal organs; for tumors whether of or behind the peritoneum, tumors of the liver, kidney or spleen; enlarged glands behind the peritoneum in the neighborhood of the mesentery of the small gut; polypi in the lumen of the gut; tumors due to appendicitis or intussusception.

In palpating, we follow a certain routine, and palpate in the region of the spleen, then over the liver, and finally in the right inguinal region (appendicitis).

**Ascites.**—The signs are the same as in the adult.

**Tympanites** gives the same signs as in the adult. In newborn infants there is in rare cases a congenital weakness of the wall of the gut. Any disturbance of the intestinal tract results in immediate distention, which may be distressing to the patient. Non-inflammatory distention is distinguished from inflammatory distention (peritonitis) by the absence of prostration or fever. There is another form of distention which precedes death in severe pneumonia or gastric enteritis. Simple tympanitic distention is seen in rachitic children in whom the lower part of the chest is narrowed and the abdomen uniformly protuberant; in these children the distention is apparent increased by the forward curvature of the spine. Percussion gives a uniformly tympanitic note all over the abdominal surface, except where feces change the note into a dullness. There is no pain except only slight general tenderness.

**Pain.**—Children may locate the pain felt in pneumonia, pleurisy or pericarditis in the abdomen. The pain may be referred to the upper part of the abdomen. The patient may complain of pain radiating to the right inguinal region, and thus in lobar pneumonia of the lower portion of the right lung mislead us into a consideration of the existence of appendicitis. In diffuse peritonitis the pain is general, but in localized disease of the appendix the limitation of pain can be made out even in young subjects. If we suspect appendicitis, it is best to examine every part of the abdomen for pain before approaching the right inguinal region.

In connection with pain and its significance, we may emphasize the fact that if the abdomen is relaxed (not retracted), showing the



grooves due to the muscular parts of the abdomen—the bellies of the recti muscles, the incurvation of the abdomen just below the border of the ribs—we may assume the absence of tympanites. In such cases peritonitis is rarely present. Pain, which has no definite localization in an abdomen relaxed as above described, may be considered as of no serious import.

The condition of the abdomen in intussusception is described in the chapter treating of that subject.

Polypoid tumors in the lumen of the ascending or descending colon may sometimes be distinctly felt in the relaxed abdomen to one side of the umbilicus.

Floating kidney in children has been recently described by Comby. The methods of examination in forms of kidney tumor or displacements of this organ are described in the chapter devoted to those subjects.

**Rectal Exploration.**—This is always carried out in the recumbent position. By rectal examination we may establish the presence of an abscess in the right inguinal region or of great swelling of the appendix in cases in which it is bound down by adhesions below the brim of the pelvis. Rectal exploration is resorted to in all cases in which we are led to suspect the presence of an intussusception. In tuberculous peritonitis also, enlarged lymph-nodes may be felt through the walls of the rectum. Kidney and ovarian tumors can in some cases be felt through the rectum.

It is not necessary to cause pain in the above procedure. On the contrary, rude examination only obscures the case. We should seek every opportunity to become familiar with the normal conditions externally and per rectum in the vicinity of the right inguinal regions in order to be able to diagnose abnormal states.

### Examination of the Joints.

Affections of the joints are among the most frequent diseases of infancy and childhood. The method of examination of the joints should be familiar to every physician. If a mother states that her baby cries when it is bathed or when it is diapered, we should examine the joints. In the newborn infant especially this holds true. If there is any limitation of motion, or should the extremities be limp, the joints should be inspected. In older children a sudden limp or intermittent obscure pain in a joint should receive attention at once.

To examine the joints, the patient should be completely undressed, and placed on a table. The spontaneous movements of the limbs are first observed before any manipulation of them is attempted. We may thus observe that one limb is favored by the infant, limitation of motion may exist, or there may be a marked

swelling of one joint. The shoulder, elbow, knee, ankle, and other joints are systematically examined. This can be done in quite short time if we make it a routine of every physical examination. In examining a joint we should not forget that when inflamed, it is very painful if not gently handled, and that any rude procedure in addition to causing pain, may injure the joint.

We first inspect the joint to see whether it is swollen, or has its normal form, or shows too plainly the prominences of the bones entering into its formation. Palpation will tell whether the temperature of the surrounding tissues is raised, whether there is fluid in the joint or whether the tissues about it are infiltrated. We also examine by mild pressure with the fingers the region of the junction of the epiphysis and diaphysis for tenderness.

Mobility is tested by flexing, extending, rotating, abducting, and adducting. During such an examination we also note muscular spasm.

**Joint-crepitus** is a peculiar crackling, rubbing sensation found frequently in the joints of infants and children. It is detected by placing the palmar surface of the hand upon the joint and moving the extremity which enters into its formation. It has been found by the writer in children who complained of no definite joint-symptoms. It may, under these conditions, be present in many joints of the same patient. Faint crepitus is found in children who have had an attack of rheumatism.

The most common affections to look for about the joints are simple luxations; syphilitic disease; osteomyelitis of a septic or infectious nature; scurvy of the joints or epiphyses in the vicinity of the joint; rheumatism, simple acute or chronic, and gonorrhoeal; tuberculous joints, especially the hip; paralysis (deltoid) of muscles about a joint; deformities, as in congenital coxa vara.

### The Spine.

**Anatomy.**—The spinal column of the newborn infant is practically devoid of natural *fixed* curves. Fehling found that there was an almost imperceptible curve backward (kyphosis) in the dorsal region and a slight lordosis in the lower lumbar region. The latter curve was more marked when the extremities of the infant were extended. The fixed curves seen in the cervical dorsal and lumbar regions later in life begin to form in the first year. They are fully fixed by the seventh year.

**Method of Examination.**—The purpose of examination is principally to discover abnormal curvatures and to test the pliability of the vertebral column. In other words, we examine for rigidity due to disease (Pott's). The patient is undressed and made to stand erect. The index finger is passed down the vertebral spinous proc-



esses, and the lines of these processes thus marked out. Any abnormal curve is thus made apparent. Painful areas are detected by pressure or tapping along the spinous processes. If deformity is

FIG. 3.



Method of testing mobility and pliability of the spine.

present, it is important to decide whether this is permanent and combined with muscular spasm (Pott's), or due to rachitis. For

FIG. 4.



Method of testing for psoas spasm.

this purpose the patient is placed on the examining table face downward. The examiner grasps both lower extremities at the ankles (Fig. 3). The palmar surface of the left hand is laid firmly on

the junction of the cervical and dorsal spine. The extremities are now raised and hyperextended with the right hand. If the spine is supple and normal, it will curve backward as the pelvis is raised toward the vertical. If there is deformity due to Pott's disease this will persist. Deformity due to rachitis will disappear under this manipulation. If the left hand is laid on the lumbar region and the above hyperextension gently carried out, first flexing the legs back at a right angle and then lifting them vertically, a distinct spasm of the muscles is felt (psoas spasm) (Fig. 4). Spinal rigidity is also made apparent by causing the child to pick up some object from the floor. Under conditions of disease the patient will hold the spine rigid in picking up the object. The hips and knees are bent but not the spine. To test the rigidity in the beginning of meningitis, the head is raised as the patient lies recumbent. In meningitis the rigidity is such that the whole trunk can be raised by placing the palm underneath the occiput and gently raising the head.

### Muscular Apparatus and Nervous System.

**Form.**—**Atrophy** of muscle is seen in any disease which affects the trophic centres of muscle in the cord. Such diseases are poliomyelitis, and neuritis following traumatism, diphtheria, measles, or any infectious disease. Atrophy is seen in joint-affections, especially about the hip. In the latter case, not only disuse, but a true reflex trophic disturbance is the cause of the atrophy.

**Hypertrophy** of muscle is seen in cases of isolated congenital hypertrophy of one limb, and also in pseudohypertrophic paralysis. In all cases of change of volume of a muscle we first inspect the affected limb and compare it with that of the opposite side if the disease is unilateral. The diseased limb is measured in its circumference and compared with the corresponding healthy limb.

**Muscle-reflexes.**—We shall take up only that aspect of the subject which should concern the practitioner in his examination of infants and children. The minutiae of electrical muscle and nerve reactions may be gleaned from works treating of such matters in detail.

The most common deep reflex is that of the patellar tendon. It is obtained by placing the infant in a recumbent position, supporting the thigh by placing the left hand beneath it, and raising it above the level of the body. When the muscles are relaxed, tap the patellar tendon sharply with the middle finger of the right hand. The procedure is similar to that employed in percussion of the chest. Both limbs are examined in the same manner. Children who can sit are placed on a table with their lower extremities dependent. When the attention of the patient is fixed upon some object tap the tendon sharply. A percussion-hammer is not necessary.



In diseases of the gray matter and of the posterior columns of the cord with trophic disturbance of the nerves (poliomyelitis, neuritis, Landry's paralysis, diphtheritic paralysis) the patellar reflex is diminished or absent.

In brain tumor and in affections of the lateral columns of the cord (multiple sclerosis, spastic disease) the reflex is increased.

The reflex is unimpaired in cerebral palsy, Friedreich's ataxia, and in cases of idiocy.

**Babinski's reflex** is a plantar phenomenon found in some forms of meningitis (tuberculous), and in diseases in which there is irritation or involvement of the pyramidal tracts. On irritating the plantar surface of the foot with the tip of the index finger there is a vigorous hyperextension of the great toe (see Fig. 70). Morse has shown that this reflex cannot be relied upon in children under the second year. I have had abundant opportunity to confirm this observation. As a differential diagnostic sign, the Babinski reflex is of little value, although I have observed it to be present more frequently in the tuberculous forms of meningitis than in the pyogenic varieties.

**Kernig's symptom** is found in children suffering from any form of meningitis and in diseases such as typhoid fever with cerebral symptoms. The sign has the same characteristics as in the adult. In infants under one year the tendency to flex the thigh in the sitting posture is normal. In these subjects, therefore, the presence or absence of this sign possesses no significance.

**Gait or Walk.**—The child is undressed, so that the feet and toes are exposed, and is caused to walk to and fro in front of the physician. The gait in disease may be ataxic, spastic, paretic, or wabbling.

**Ataxic gait** is seen in children suffering from Friedreich's ataxia, or from tumor involving the motor centres for the lower extremities. The gait is uncertain; patients walk as if inebriated, with the feet wide apart. Incoördination of movement is characteristic of all these cases. We must in all cases distinguish between simple muscular weakness, as in pseudohypertrophic paralysis, and convalescence from acute disease, such as fevers, and a weakness combined with a palpable defect in the power of coördinate action. In cases of cerebral disease, as a rule, there is lack of coördination elsewhere, as in the muscles of the upper extremities. In these cases the coördination is tested in older children by telling the patient to close the eyes, and directing him to touch the tip of the nose with the index finger of the right hand several times in succession. In cases of ataxia there will be great uncertainty in carrying out this manœuvre. In diphtheric paralysis there may be combined with a real weakness, ataxia or incoördinate movement. If we remember that in these cases there is a neuritis, with consequent

atrophy of muscle and loss of reflex, we shall not commit the error of overlooking the paralysis in our desire to account for the condition present as a simple muscular weakness the result of the illness. In these cases there may also be paralyzes of the trunk muscles causing inability to assume the upright posture. In ataxia caused by cerebral tumor there is in certain cases a crossed hemiplegia (pons tumor), with foot-clonus and paralysis of ocular muscles which aid in the diagnosis.

**Cerebellar Titubation.**—In cerebellar tumor, which is the variety most common in children, there are at the outset, in most cases, disturbances of the gait or ataxia. The patients walk in an uncertain manner, generally staggering to one side. In severe forms of this disease the patients will fall to one side if not protected. The cases thus far recorded all show early involvement of the optic, auditory, and other cranial nerves, abducens paralysis, with symptoms of vertigo.

**Spastic Walk.**—This walk is so characteristic as not to be easily mistaken for anything else. It is found in all forms of spastic paraplegia, congenital or acquired. There is not only actual spasm but also weakness of muscle. There are other phenomena of nervous disturbance, such as increased patellar reflex and foot-clonus. The patient seems to drag his legs in walking. Each extremity is brought rigidly forward, the toes scraping the ground. The muscles may or may not be well nourished. Electrical contractility may or may not be increased. The children may walk cross-legged (Gowers). At first there is inability to walk; later in childhood locomotion is possible. In certain forms the spasm of the extremities is so great as to keep them in constant extension at the knee; flexion in these cases can only be attained with great expenditure of force.

In infants and children who cannot walk and are the subjects of spastic paraplegia the characteristic position of the lower extremities may be made apparent by supporting the patient on the feet. In all of these cases, as soon as the toes touch the ground the reflex produces the characteristic extension of the limbs, with the toes on the ball of the foot on the ground and the heel raised.

In very young infants who are the subjects of amaurotic idiocy the spastic phenomena are sometimes very marked. In these cases there are other symptoms, such as amaurosis and inability to hold the head upright; the presence of the Tay-Kingdon spot in the fundus of the eye aid in the diagnosis.

**Limping Gait.**—Joint-affections cause simply a limping gait; a study of the joint, as described elsewhere, will aid the diagnosis.

**Infantile paralysis**, or cerebral palsy, at the outset causes the characteristic dragging of the extremity if the paralysis is not complete. Infants in whom there is a complete loss of power in one of



both lower extremities give a history as follows: The infant may have been able to walk or stand; the attack suddenly deprives it of the power of motion. There is a limp extremity on one or the other side, with rapid atrophy of muscle and loss of reflex. In cerebral palsy there is no atrophy and the tendon reflex is present.

The methods of examining the mouth and special organs will be taken up in the chapters devoted to their diseases.

### THE ADMINISTRATION OF DRUGS, AND OTHER METHODS OF THERAPY.

Children should receive drugs in an agreeable form, although some may take nauseous drugs with apparent indifference. Bulky mixtures or drugs which are apt to upset the stomach should not be prescribed. The author recently saw a severe enterocolitis set up by a cough mixture containing antimony. Drugs should not be administered in pill form to infants or children. Tablets are a ready means of administering certain drugs. They can be crushed and given in a teaspoonful of some indifferent fluid. Powders are also easily taken. They are put in a spoon, some fluid added to form a mixture, which is then administered. Quinine is given either in syrup of yerba santa or in chocolate powder and water; or the child is given a piece of chocolate to eat, and then is caused to take the quinine immediately afterward. A child should never be forced to take a medicine. Much harm is done in this way. Certain drugs, such as opium in the form of the simple tincture or morphine, are not given to children under the age of two years. Atropine, of late advocated in cholera infantum, should not be given to infants and young children. They bear this drug badly. Jaborandi is badly borne, as is also apomorphine. Camphor is a very good cardiac stimulant. It is useful in collapse, but must be given cautiously in cases in which there is diarrhoea. In the latter disease the camphor is apt to irritate the stomach and gut. The coal-tar series, such as antipyrin, antifebrin, and phenacetin, are powerful depressants. In those cases of fever in which it is not possible to give baths to lower the temperature we are sometimes forced to administer these drugs. It is then well to combine with them some caffeine.

If a child or an infant refuses to take a drug, it may be put in a teaspoon, the spoon held horizontally to the lips, and when the mouth is opened the spoon carried far back into the mouth and tilted. The spoon is held in the mouth until the act of swallowing, which must inevitably take place, is completed; the spoon is then withdrawn. If this manœuvre is thus carried out, the fluid will not

be rejected. Holding the nostril closed, and thus forcing the child to open the mouth, is bad practice. Patience and suasion can accomplish as much in most cases.

Digitalis is not given continuously, but is administered for one or three days, and when the pulse begins to show signs of less frequency its administration is suspended. Alcohol is well borne by children. I do not hesitate to administer it in cases of nephritis if the heart is weak. In the gastro-enteritis of nurslings the stomach is very intolerant of alcohol. It should not be given even in very severe cases with great prostration, as the vomiting is apt to be aggravated. Much has been written concerning antipyresis and antipyretics in the treatment of the diseases of infancy and childhood. The young practitioner can feel assured that high temperatures are well borne by infants and children. A temperature of  $106.5^{\circ}$  F. ( $41.3^{\circ}$  C.) in an adult, although of short duration, would cause great alarm, and rightly so. On the other hand, such a temperature in an infant or child does not necessarily threaten life, as it is incompatible with recovery. A convulsion in some children is the direct result of a rise of temperature. Such a convulsion does not necessarily lead to others nor to epilepsy. The heart and kidneys bear long-continued high temperature well in comparison with those of the adult. The most trivial causes will cause a rise of a degree or two in the temperature of an infant or a child. Taking all the idiosyncrasies into consideration, it may easily be understood to the student and young practitioner why it is essential that methods of therapy should be modified before they can be applied to infants and children. A reduction of temperature from  $104^{\circ}$  to  $102^{\circ}$  even if it can be accomplished by a coal-tar derivative, does not cure the patient. Some diseases, such as measles, scarlet fever, pneumonia, and a host of others, run a course of high and continued temperatures extending over a certain space of time. If an infant or child is attacked with convulsions following every acute rise of temperature, the parents should be warned of this fact. In these cases, as soon as a rise of temperature is noted, it should be combated by every means in our power. Reduction of temperature in such children at the outset of a disease is of the highest utility. It saves the nervous system from the shock of a convulsion. Hydrotherapy is, as a rule, the safest and most satisfactory antipyretic measure at our disposal.

The dosage of drugs for infants and children has received much attention. In practice we judge more by the action of a remedy than the quantity administered. The initial dose should be small. Infants under a year receive  $\frac{1}{20}$ th of the adult dose, and at the end of one year  $\frac{1}{10}$ th of the adult dose is safe. At the fifth year  $\frac{1}{5}$ th and at the tenth year  $\frac{1}{2}$  the adult dose is the rule. These figures are not absolute. Nitroglycerin if given in doses of less than



$\frac{1}{250}$ th of a grain has scarcely any effect on children five years of age. On the other hand, strychnine may be safely given in quantities of  $\frac{1}{250}$ th of a grain to infants, and  $\frac{1}{150}$ th of a grain to children two to three years of age. It will be seen that if the hard-and-fast rule of division of doses according to age were followed, these drugs would necessarily be given in much smaller dose, and their action would be correspondingly inefficient.

Hypodermic administration of drugs to infants and children presents nothing peculiar, as compared with the same method applied to adults.

### Hydrotherapy.

The practice of hydrotherapy as applied to the adult must be somewhat modified before it can be carried out with the infant or the child. The reason for this is that the infant or child does not react so readily and cannot bear sudden changes of temperature so well as the adult.

**The Sponge Bath.**—A rubber sheet is placed on the crib, and over this one layer of a small blanket; the patient is then placed nude on this blanket and covered with another blanket. There is thus no undue exposure. A small basin of water at 80° to 85° F., with a dash of alcohol, is now brought alongside of the crib. With a small sponge or piece of soft folded linen the parts of the patient are sponged; first one arm, then the other, then the trunk, and finally the lower extremities. As each part is exposed, the rest of the body is kept covered. This procedure is repeated until the body has been sponged for five or ten minutes. This method of hydrotherapy is especially suitable in acute rises of temperature of short duration and in mild cases of continued fever in which the temperature does not rise high.

**Cold Chest Compress.**—Three layers of linen are cut so that they will envelop the trunk from the clavicles to the umbilicus. The general shape should be that of a shirt deprived of arms and open at the sides. On the outside of this linen compress there should be a compress of Shaker flannel cut in a similar manner. The compress of linen is moistened with water at 80° to 85° F. With robust children the water may be at 70° F. The compress is wrung out and applied so that the neck, shoulders, and chest are covered as with a shirt. The flannel is now applied to the outside. The compress is moistened every hour with water at 70° to 85° F. and re-covered with the flannel.

The **cold pack** is not so useful in the treatment of the febrile conditions of childhood. The method is similar to that followed with the adult, with the exception that the sheet is moistened with water at 80° to 85° F. In other cases the patient, after being wrapped in such a sheet, is rubbed by the attendant with ice on the

outside of the sheet. The author has had no extensive experience with this method.

**The Full Bath.**—The full bath, as advocated by Brand, is seldom carried out in the treatment of children. Children struggle against the bath, and if the temperature is too low, they become so depressed that it is difficult to rouse them. I therefore place children with typhoid fever, or pneumonia, or scarlet fever in a bath at  $100^{\circ}$  to  $105^{\circ}$  F., and lower the temperature to  $80^{\circ}$  or  $85^{\circ}$  F., applying friction to the body constantly. After five or ten minutes the patients are taken out of the bath and rubbed dry. Warm-water bottles are applied to hands and feet.

In conditions of delirium and coma with a high temperature, in which the heart is weak, I have given baths at a temperature of  $105^{\circ}$  to  $108^{\circ}$  F. The cases in which these baths are indicated are those in which any application of cold water causes cyanosis and collapse. I have seen infants suffering from bronchopneumonia, with high temperatures, in a condition resembling a rigor after a bath at  $85^{\circ}$  F. With these infants the warm bath acts as a cardiac stimulant and quiets the nervous symptoms.

### Hypodermoclysis.

Hypodermoclysis is the injection into the subcutaneous tissue of either a 0.6 per cent. salt solution or the normal salt solution of Cantani (sodium chloride, 4 parts; sodium carbonate, 3 parts; water, 1000 parts). It is indicated in infants suffering from cholera infantum and in other exhausting states. Monti, who was the first to apply this mode of therapy to the infant, injects 100 to 200 c.c. at a time. Epstein showed that smaller quantities—10 to 40 c.c.—are more beneficial and more quickly absorbed. Experience teaches that large quantities of fluid injected subcutaneously cause extensive blood extravasations in exhausted infants and much subsequent pain. The solutions used should be freshly prepared and sterilized. Welch has reported cases of infection with *Bacillus aerogenes capsulatus* following hypodermoclysis. I have had one case, although every precaution was taken to avoid infection.

A large antitoxin syringe, holding 20 c.c., is used. It should be carefully sterilized.

From 20 to 30 c.c. of the solution are injected two or three times daily into the subcutaneous tissue of the lumbar region or abdomen. Monti injects into the subcutaneous tissue of the abdomen. Massage should not be performed after injection, as it is very painful and causes hemorrhages. The puncture wound is covered with a piece of sterile gauze. The main point is to inject small quantities of the solution at intervals of from four to six hours, and watch the effect. The action is that of a stimulant to the heart and the



processes of resorption. Epstein showed that within a few hours after injection of salt solution the proportion of hæmoglobin and red blood-cells was reduced. As salt solution has a dissolving effect on the red blood-cells, the injection of large quantities of the solution may be harmful.

### Syringing the Nose.

**Instruments.**—The best form of syringe for this purpose is an olive-tipped glass syringe. Some forms are made with a soft-rubber tip. The tip should be blunt, lest the nares be injured (Fig. 5).



FIG. 5.

Nasal syringe. Correct shape.

FIG. 6.



Method of syringing the nose in the upright posture.

The solution used is generally a normal salt solution.

**Method.**—The infant or child is wrapped in a sheet or blanket,

and held in the lap of a nurse, who holds a pus basin beneath chin. The operator stands behind the patient. The syringe is horizontally to the floor of the nares and the solution slowly injected into the nostril (Fig. 6). If successfully performed, the process results in the solution's coming out of the other nostril. There is no danger in the manoeuvre if carefully carried out. If the infant is too weak, the nares may be syringed with the patient in the recumbent posture. The nurse stands at one side, and the infant is placed on the side, the pus basin beneath the nose, as shown in Fig. 7. A rubber fountain-syringe may be used in the same manner.

FIG. 7.



Method of syringing the nose in the recumbent posture.

Here also the position of the syringe is horizontal to the floor of the nares. The syringe should be thoroughly boiled before and after using. An old syringe should never be used, no matter how carefully it has been sterilized.

### Vapor Spray ; Calomel Inhalations in Acute Laryngeal Disease

With infants and children the spray is not so useful an agent as steam vapor impregnated with balsams or turpentine, and combined at times with inhalations of the fumes of sublimed calomel. The spray cannot, as a rule, be used locally except with the most tractable children. With infants its use is not feasible.

The vapor of steam impregnated with balsams or turpentine is very useful in all forms of acute laryngitis in which there is bronchitis. I dispense with steam vapor if bronchitis is present. The mode of application in catarrhal or membranous croup is as follows: The crib is covered with a sheet suspended from the



upright poles fastened to the corners of the crib. A tent is thus formed. The croup kettle is placed at one side of the crib, in such a manner that the steam vapor escapes into the improvised tent. The vapor is medicated by placing in the kettle a teaspoonful of turpentine or thymol. This will be readily vaporized. No special apparatus has any advantage over the ordinary croup kettle. If calomel sublimations are to be given, they should be combined with the steam vapor. Ten grains of calomel are placed in a spoon held over an ordinary candle, and the fumes led under the tent, the air of which is impregnated with steam vapor. The special devices sold for the sublimation of calomel may be used, but possess no advantage over the method described above (Fig. 8). Calomel sublimations are exceedingly irritating, but they relieve the patient very promptly. They may be continued for forty-eight hours at intervals of two hours, without fear of salivation.

FIG. 8.



Sublimator for calomel inhalation.

### Stomach Washing.

One of the most valuable additions to our therapeutic armament within recent years is stomach washing in case of the nursing infant. No improvement has been made upon the method as first proposed by Epstein. The cases in which it is indicated are mentioned in another part of this work. The procedure is easiest of application to nurslings in whom there are no teeth or in whom very few teeth have erupted. With these subjects there is no danger of the catheter's being bitten, and there is no necessity of using a gag. With older children, however, a gag must be used when stomach washing is attempted. The Denhardt gag of the O'Dwyer set of intubating instruments is most suitable for this purpose.

**Indications.**—Washing out the stomach is principally indicated in the acute gastro-enteritis of the summer months. It is not bottle-fed infants alone that are attacked, but even breast-fed infants may be thus affected. The winter months also furnish their quota of these cases. One vomiting spell, as it is called, does not require attention. If, however, on suspension of the bottle or breast, vomiting continues and becomes uncontrollable, we proceed to stomach irrigation. Another indication is the so-called chronic dyspeptic

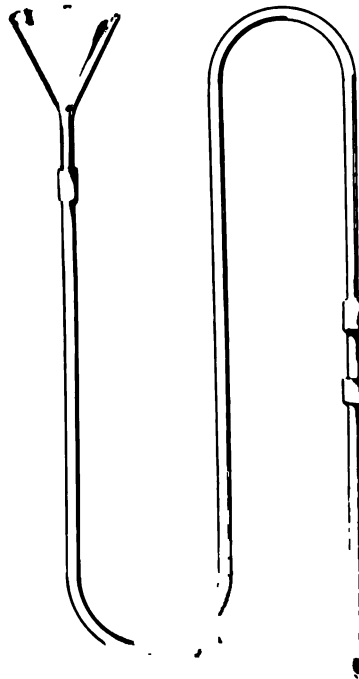
vomiting. Those who advocate this method of treatment ~~must~~ ~~never~~ forget that, above all, the food is at fault, and must be ~~and~~ modified. I do not favor washing the stomach in the

One washing is, as a rule, sufficient. I have rarely repeat it. If vomiting persists after the first washing, it looks for other conditions than gastro-enteritis as the cause vomiting, such as intussusception.

Acute drug poisoning or ingestion of any irritating fluid relieved by stomach washing. I have washed out many who had been given an overdose of paregoric, or who had put in green, turpentine, or other drug. If, as sometimes I child accidentally swallows a caustic alkali, we should not the tube into the oesophagus or stomach.

**Method.** A four-ounce funnel, a piece of rubber tubing

FIG. 9.



Apparatus for washing out the stomach

a half size larger, and a No. 14 rubber catheter are the necessary. The rubber tubing is attached to the funnel mouth, or a piece of glass tubing to the catheter, is. About a quart of normal saline solution is needed. Then



PLATE I.





of the water should be at least 100° F. The operator needs one assistant. The infant is completely undressed, and is then wrapped in a blanket, the diaper having first been applied. The hands are tucked in with safety-pins. The infant having been laid recumbent on a table, the operator, standing on the right, introduces his left index finger into the mouth and depresses the tongue (Plate I.). The catheter, moistened with water, is now introduced and passed backward. With gentle urging the catheter passes easily into the oesophagus. There is no likelihood of the catheter's passing into the larynx and trachea. About six inches of the catheter are introduced. The funnel is depressed and the stomach contents are first allowed to flow out. The funnel is then raised about two feet above the patient, and the assistant slowly pours the saline solution into the funnel, the fluid flowing into the stomach. Before the funnel is completely emptied, it is lowered and the stomach contents siphoned out. This operation is repeated several times, until the water returns quite clear. If during the stomach washing the fluid should be ejected from the stomach in the act of vomiting, it will easily flow out of the mouth if the infant is recumbent. There is not the slightest danger of aspiration of the fluid into the trachea. I think the recumbent position is superior to the sitting posture advocated by some clinicians. A young infant is unable to sit up of its own accord. The introduction of the tube is not so easy for the infant in the sitting posture as in the recumbent position. The tube being introduced, the stomach contents sometimes refuse to flow out because mucus and food particles obstruct the lumen of the catheter. In such cases the catheter is withdrawn, and washed out. The catheter is then pinched with the fingers in such a manner that some of the water or washing solution remains in the catheter. It is then introduced into the stomach. In this way the catheter, being filled with fluid, mucus and food cannot obstruct the lumen of the tube before siphonage is begun. Fluid can then readily be introduced into the stomach. These difficulties occur in cases in which there is a large amount of mucus in the stomach. The finger should always be retained in the mouth. By grasping the catheter with the thumb and index finger of the right hand, prying open the mouth at the same time, we prevent pressure on the catheter during the washing. If the infant has upper and lower incisors, the catheter must be held at one side of the mouth and the mouth kept open by means of the index finger held in the angle of the mouth. The method described above has been followed by me for years. I have never had an accident.

#### Gavage.

Gavage is a method of forced feeding by means of the stomach-tube. I have not practised this method of feeding infants. Older

children suffering from pneumonia or typhoid fever, and delirious or unconscious, have been fed with success by this method inwards.

The method of procedure is similar to that used in stomach washing. It is best not to introduce the catheter through the nose, to keep the mouth open with some device. If the catheter is passed through the nose, no food should be introduced into the funnel until we are sure the feeding-tube is in the stomach. With older children a tube passed through the nose may pass into the larynx. If it has done so, a hissing sound will be heard. Aphonia will also be present. In infants and young children the glottis is small, and a full-sized catheter will not readily pass into it. After the tube is in the stomach the prescribed amount of liquid food is introduced and the tube rapidly withdrawn. The feeding may be repeated every four to six hours.

### **Rectal Enemata; Irrigation; Enteroclysis.**

The bulk of an ordinary enema, introduced in order to empty the bowel, should be from 2 to 4 ounces. A Davidson's bulb syringe should not be used. A No. 16 or No. 18 catheter is attached to the nozzle of an ordinary four-ounce hard-rubber syringe. The infant or child is placed on its side, with a rubber sheet under the buttock. The tip of the catheter is oiled and passed well within the anal ring. The catheter is then attached to the nozzle of the syringe containing the fluid to be injected, and the fluid is gently thrown into the rectum. An enema commonly used is soap-water with the addition of a tablespoonful of castor oil or glycerin.

The high rectal enema, irrigation, or enteroclysis, is given in all forms of summer diarrhoea, dysentery, and in typhoid fever. It is also indicated in cases in which there are symptoms of collapse, in exhausting diseases, in nephritis, and after operations. It is also used to reduce intussusception. In diarrhoea, the object of the high rectal enema is twofold—to clear out the feces from the lower bowel, and to supply fluid to the depleted circulating blood, thereby stimulating the heart. The latter is the main object in practising enteroclysis in states of exhaustion and after operations. In suppression of urine we aim to supply fluid to the kidneys and stimulate the circulation. According to Kemp, the high rectal enema is one of our most useful diuretics.

The solution employed is the Cantani saline solution (sodium carbonate, 3.0; sodium chloride, 4.0; water, 1000). At least a quart is injected. The temperature of the solution for simple washing of the gut, as in diarrhoea, should be that of the body. In nephritis or collapse the temperature should be at least 108° to 110° F. (42.2° to 43.3° C.).





PLATE II.



The instrument employed may be a bag fountain syringe, of a quart capacity, to which is attached a small calibre soft-rubber rectal tube or a catheter, or the rubber tubing and catheter may be attached to a six-ounce glass funnel.

The patient is completely undressed and laid on a table on the side, with the knees flexed and the buttocks near the edge. A rubber sheet placed underneath the buttocks leads into a pail, so that the returning water will drain off (Plate II.). The buttocks are placed slightly higher than the trunk. The catheter or rectal tube is oiled and introduced two or three inches into the rectum, the water allowed to flow, and the tube passed higher up. Sometimes there is an obstruction to the passage of the tube, and then it is necessary to introduce the finger cautiously into the rectum alongside of the tube and guide it past the upper sigmoid ring. The tube may thus be passed from six to eight inches into the gut. It is seldom necessary to introduce it higher, as the water will find its way into the colon. About a pint or more of water is then allowed to flow into the gut. It is not necessary to compress the anus around the catheter to prevent escape of the fluid. Some of the fluid may escape alongside the catheter. In some forms of exhausting diarrhoea a portion of the saline solution should be left in the gut after it has been well irrigated, in order to stimulate the heart and supply fluid to the circulation. Two irrigations may be necessary in the twenty-four hours, rarely more. In typhoid fever one low irrigation is given daily. In some subjects, if the irrigations are continued too long, hyperæmia of the mucous membrane results. Clinically, this is manifested by a continuance or increase of mucus in the washings, and also by the occasional presence of blood. In such cases the enemata must be suspended. In nephritis complicating scarlet fever, rectal irrigation is one of the recognized methods of stimulating the secretion of the kidney, which result, according to Kemp, begins twenty minutes after the fluid is introduced into the gut. With adults the Kemp tube is used, but with children, who are difficult to keep quiet, continuous irrigation is not feasible. In these cases high enteroclysis is given in the ordinary manner, as much of the solution as possible being retained in the rectum. This procedure may be repeated two or three times daily. In giving ordinary enteroclysis the bag of the fountain syringe or funnel should not be held more than three feet above the body of the patient, lest the pressure be too great. About a pint of fluid at a time is allowed to flow into the gut; the catheter is then disconnected, and the contents of the gut allowed to flow out.

A stimulating enema is given after an operation, or when symptoms of collapse appear in any acute illness. Only small quantities of solution are allowed to flow into the rectum. A formula in use in my wards is the following :



Whiskey . . . . .	℥j.
Caffeine . . . . .	gr. ʒ.
Tinct. digital. . . . .	gtt. ij.
Sol. sodium chloride (0.6 per cent.) . . . . .	℥j.
Temperature, 102°-105° F.	

Nutritive enemata are used when for any reason, such as uncontrollable vomiting, the stomach must be given complete rest. Some touse solution, ℥j at a time, is given every four hours. Or, ex pancreatis, gr. v; sod. bicarb., gr. ij; water, ℥iv; milk, ℥xvj; with or without the addition of an egg. Give ℥j or ℥ij. These enemata should be given slowly and high up, and in small quantities at a time.

For constipation the following is excellent:

Olive oil . . . . .	℥ij.
Glycerin . . . . .	℥j.

### HYGIENE OF INFANCY AND CHILDHOOD.

**The First Bath.**—The temperature of the room in which the newborn infant is bathed should be from 70° to 72° F. The bath should be given near an open fire or a stove, in order that the infant may not be chilled. To remove the vernix caseosa, the body is anointed with olive oil, vaseline, or benzoinated lard; of these vaseline is more irritating than olive oil. In anointing the body only a portion of the surface is exposed at a time. The object of the bath is to remove bacteria or substances which may decompose on the surface of the body. The washing should be done rapidly and thoroughly. The temperature of the water used should be 100° F. and not 96° F., as recommended by some. The baby is taken from the bath, placed in a dry, warmed towel, and carefully dried. The skin must not be rubbed too harshly or unduly stretched, especially in the groin and axillæ. If roughly handled, the skin in these regions will crack and fissures will be produced.

The **cord** is dressed with absorbent, sterilized gauze. The gauze is folded four times into a pad three by four inches, and a hole for the passage of the stump of the cord is cut through the centre. The gauze is then folded over the stump, the whole laid flat against the abdomen, and the binder applied. This dressing is changed daily.

**The Eyes.**—Immediately after the first bath a drop of a 2 per cent. solution of silver nitrate is dropped into each eye. This is a prophylactic measure against gonorrhœal ophthalmia. The eyes are washed once daily with lukewarm water to which a pinch of salt has been added.

**The Daily Bath.**—The infant should be bathed daily in the forenoon, one hour after nursing. The temperature of the water

may vary from 96° to 98° F. during the early months; after the sixth month the temperature may be as low as 90° to 92° F. Cold baths and cold sponging are not beneficial for infants. The first bath reduces the temperature, so that the normal status is not regained for a period of from one to three days. During early infancy the temperature of the bath should not be lowered. Reduction of the temperature of the bath or of the room distinctly retards increase in weight. Moreover, we should not attempt in this way to "harden" the baby. Some infants may be unduly chilled during any bath. For this reason the precautions mentioned in the section on the Newborn Baby's Bath must be observed.

**The temperature of the room** in which an infant sleeps should be at least 70° F. The room should be well ventilated and free from draughts. An open fireplace with a log fire is the most hygienic method of ventilating a nursery. As stated above, the infant must be protected from wide variations of temperature, in order that its vitality may not be reduced, and the increase of body-weight may progress in a physiological manner.

**The Body Binder.**—The binder should be of soft, thin, white Shaker flannel, five inches wide and sufficiently long to pass two or three times round the body. It is secured with strings, not with pins. It is useful at first in retaining the dressing of the cord in place, and later on in supporting the umbilicus during straining and attacks of crying. The binder is discarded when the infant first attempts to stand—usually at about the seventh month.

**Clothing.**—The clothing of the infant should consist of a chemise of wool next the skin, and over this should be a loose garment, also of wool or flannel, reaching from the shoulders to below the feet, and sufficiently long to allow of being folded upward. Garments should not constrict the chest. The chemise, which should be of gauze weight in summer, is worn both winter and summer. In some infants contact of wool with the skin causes an eruption of sudamina. This may be avoided by placing a fine linen chemise next the skin, and over this the gauze wool garment.

**The Skin.**—The precautions to be observed in drying the skin have already been mentioned. Dusting powder is best applied with a puff of absorbent cotton. This can be thrown away and a new one used at each dressing. In order to prevent caking, any excess of dusting powder should be removed.

If the skin is subject to sudamina in the summer, the bath should contain bran. A handful is added to the water, or the bran is put into a gauze bag and this is placed in the water; when well soaked the bran is expressed into the bath. Salt water irritates a skin which is the seat of sudamina.

**The Mouth.**—It was formerly customary to wash the mouth of the infant thoroughly twice or thrice daily. This is no longer done.



The mouth of the breast-fed infant should not be washed. If the breast nipple is kept scrupulously clean, sprue or stomatitis will be avoided. The nipple of the nurse's or mother's breast should be cleansed with a solution of boric acid before and after each nursing. Before the eruption of the teeth the natural secretions of the mouth are quite sufficient to keep the mouth clean. After the teeth appear, they are kept clean by washing them gently twice a day with a piece of lint wet with boric acid solution. With bottle-fed infants the procedure is somewhat different. The mouth is cleansed once a day by means of a piece of lint and warm water or solution of boric acid. The utmost gentleness should be used. The roof of the mouth and the hamular processes of the palate bones are avoided, since friction in these localities will invariably cause ulceration (Bednar's aphthæ). The fingers and the nails should be carefully cleansed before introducing them into the mouth. The bottle nipples should be boiled in water once a day for fifteen or twenty minutes, and cleansed with hot water after each nursing. In the intervals of nursing the nipple is kept wrapped in a piece of clean absorbent gauze. In this way sprue will be avoided.

**The Diaper.**—After each movement the infant is gently cleansed with a piece of lint or old washed muslin and water. A sponge should not be used. The parts are dusted and the excess of powder blown away. The bulk of the feces is removed from the diapers, which are then allowed to soak either in a weak solution of carbolic acid (1:1000) or in sublimate solution before being placed in the wash. After changing the diapers the nurse's hands and nails should be scrupulously cleansed with brush and file. This cleanliness of the hands is important even with breast-fed infants, since in this way contamination of the infant's food with fecal bacteria is avoided. Diapers are boiled in water to which soda has been added. A clean diaper should be aired and warmed before being applied to the infant.

**Temperature.**—The temperature of infants is always taken in the rectum; that of older children is also taken in the rectum when possible. In some children there is an innate modesty which forbids the use of the rectum for this purpose; in these subjects the temperature should be taken in the mouth or axilla. All patients should have thermometers of their own.

In the newborn infant the temperature will vary from 36.9° to 38.4° C. (98.4° to 101.1° F.); the latter is exceptional. According to the studies of Lachs, the average temperature of the newborn infant varies from 37.5° to 37.9° C. (99.5° to 100.2° F.).

After the first bath the temperature falls from one and a half to two and one-tenth degrees F. Two hours after the bath the temperature begins to rise, and reaches its original height within twenty-four hours, sometimes later. In premature and weakly infants the temperature does not reach the original figure for fully three days,

and in some infants it may never reach the original height. The body temperature of infants shows slight fluctuations during the day. The maximum temperature is in most cases reached at mid-day and afternoon; the minimum during the morning and evening. The daily fluctuations vary from one-tenth to three-tenths of a degree F. During sleep the temperature sinks from three-tenths to one-half a degree F. (Alix, Vierordt). In general, we may say that in infants and children any temperature from 99.3° to 100° F. in the rectum is normal.

The following table of body temperatures is compiled from Lachs, Vierordt, Alix (rectal):

Newborn infant. . . . .	37.5° to 37.9° C. ; 99.5° to 100.2° F.
5-16 months . . . . .	37.4° to 37.9° C. ; 99.3° to 100.2° F.
20 months-4 years . . . . .	37.5° to 37.9° C. ; 99.5° to 100.2° F.
5-9 years . . . . .	37.6° to 37.8° C. ; 99.6° to 100.1° F.

**The Breasts of the Newborn Infant.**—From the third to the fifth day after birth milk appears in the breasts of both sexes. As

FIG. 10.



Caking of the milk in both breasts of a newborn infant.

a rule, the secretion appears earlier in girls. The breasts may become swollen and tense. Sometimes one gland, generally the right, functionates sooner than the other. The secretion has been



examined by Barfurth, Herz, and others, and found to be composed of proteids, 2.5 to 3.6 per cent.; fat, 2.5 to 3 per cent.; and sugar, 2.5 per cent. It is a secretion of milk. The method of secretion is the same as in the adult gland. The amount of milk, which is called by the laity "Hexen milch" (Ger.) or witches' milk, is small. The breasts should be kept scrupulously clean. If they become tense, they should not be rubbed or manipulated. We cannot bandage these breasts, as in the adult, for the thorax being resilient, the bandage may interfere with the respiratory movements and thus cause serious pulmonary trouble. The secretion of milk lasts, as a rule, from six to eight weeks; in exceptional cases it may continue six months (Herz). Mastitis is the result of infection, and not of caking of the breasts. If actual caking occurs, it is permissible to massage the breast gently once a day with sterilized oil. The index finger is moistened with a drop of the oil and the gland is gently stroked in a circular direction for five minutes. Before performing this office the breasts and the finger should be scrupulously cleaned to avoid infection. Cold applications or applications of ointments are to be avoided. If the breasts are soft, even though tense, they should be let alone.

**Open Air.**—An infant may be taken into the open air two weeks after birth in summer, and three weeks after birth in winter. After this a daily open-air exposure is allowable in good weather. If the infant is warmly clad in winter, there is no danger. A veil should be worn in order to protect the skin of the face from the irritation of dust. A delicate skin thus protected will not become eczematous. Two hours in the open air in the forenoon and two in the afternoon are sufficient. The infant should be indoors after 4 P. M.

**Sight.**—According to Preyer, the infant during the first month will awaken if a bright light is suddenly flashed in its face. At the seventh month it distinguishes objects apart.

**Hearing.**—A baby does not hear in the true sense of the word until the beginning of the second month. After this, hearing develops, and loud talking and noise will disturb it. At the ninth month the infant attempts to articulate and talk.

**Standing and Walking.**—The infant will try to stand in the arms of the nurse or mother at the seventh month. At the tenth month it will stand without aid. At the eleventh month it will attempt to walk if aided. At the sixteenth month it will walk without aid. The infant should not be allowed to sit up until the seventh month, at which time its attempts to stand are evident.

### NATURAL FEEDING OF INFANTS AND CHILDREN.

Although many infants can be successfully reared with our improved methods of substitute feeding, the breast-fed infant is in the end



PLATE III.

FIG. 1.



Form of the Breasts of a Wet-nurse with Abundant Milk of Good Quality. (After Schlichter.)

FIG. 2.



Form of the Breasts of a Wet-nurse whose Milk is Deficient in Quantity and Quality. (After Schlichter.)



better fitted to enter upon the struggle for existence than one fed on the bottle. No matter how skilfully artificial food may be prepared, it will not in some cases be assimilated. Great difficulty is experienced in feeding premature infants with the bottle. Some infants, when deprived of the breast, suffer from colic and have green curdled movements, are restless, and cry all night; others have a moderate amount of colic, and the movements are yellow and contain whitish curds. Such infants lose ground steadily, or remain stationary in weight. They should not be kept on the bottle; they will not thrive no matter how we may modify the milk. There are other considerations which make it desirable that the infant should, if possible, take the breast. Even if the milk is insufficient in quantity, it is a mistake to reject it wholly. In such cases the baby should be fed on the breast, aided by the bottle. Finally, in spite of the disadvantages of placing a baby at the breast of a stranger, both from a moral and an economic standpoint, we should endeavor to feed every infant on breast milk. Failing in this, we must have resort to a substitute.

#### **Selection of a Wet-nurse.**

It is not necessary that the wet-nurse should have been recently delivered. A newly born baby may be given the breast of a nurse whose baby is from one to two months of age. In fact, her milk is preferable to that of a nurse who has just been confined. For, apart from the uncertainty as to whether the milk will agree with the baby, the milk after a few weeks attains a uniform constitution, and is more likely to agree with the baby than milk from the breast of a woman recently confined. I prefer to place the newly born infant on a breast at least three weeks old.

The method of examining a wet-nurse as to her fitness begins with ascertaining the history of her own baby. It should sleep well in the intervals of nursing, be free from colic, and have normal movements. The baby should be completely undressed for examination. It should be at least tolerably well nourished. No eruption should be visible on its skin. There should be no copper-colored intertrigo, no snuffles, no pigmented spots, and no rhagades around the mouth or anus. The head should not have an idiotic, microcephalic conformity. The wet-nurse should be below the age of thirty. Old multiparæ do not, as a rule, furnish good milk. The shape of the breast is important. The pear-shaped, elongated, hanging breast furnishes more milk than the firm round breast of virgin shape (Plate III.). The nipple should be about one centimetre long and three-fourths of a centimetre in diameter. The baby can easily grasp such a nipple and draw it into the mouth. A flat nipple, or a nipple with fissures, or a nipple surrounded by eczema, is not desir-



able in a nurse, and may even be dangerous to an infant. The nurse is next directed to undress, and her body is examined for traces of any eruption which may be specific. Pigmented macules should arouse suspicion. The lungs, especially the apices, are examined for bronchitis or tuberculosis. The nurse is rejected if there be the slightest evidence of apical involvement. The teeth should not be carious to such an extent as to preclude their being kept clean. The presence of a fetid ozæna is highly objectionable, apart from the offensive odor. Such cases may be latently tuberculous. The woman should be mentally sound. The wet-nurse is then examined as to the presence of venereal disease by inspection of the introitus vaginae and the anus. Search is made for mucous patches and suspicious cicatrices. After having examined both child and mother in the manner detailed, we are in a position to recommend the nurse if the milk is satisfactory.

The physician should have at hand in his office means by which he can at once decide upon the desirability of a wet-nurse. He must not at the beginning be driven to the necessity of a milk analysis. He decides first as to the quantity and then as to the quality of the milk. As a rule, a wet-nurse comes to the physician insufficiently fed and in a frame of mind far from tranquil. If despite these conditions the milk possess the qualities desired, he may at once venture to place the baby at the breast. If the milk does not agree with the baby after a fair trial, future conduct will be guided by certain developments, both in the quantity and quality of the milk and the condition of the infant.

**Quantity of the Milk.**—The physician grasps the breast in the palm of his right hand and gently but firmly attempts to express the milk. The milk should with gentle pressure flow freely from the ducts. A drop is caught on the nail of the thumb. This time-honored nail-test is not to be despised. A drop of good milk will retain its bluish-white tint. This test will bring out the color of the milk, whether too watery, yellow, or white, to the experienced eye. The nurse is then directed to pump by gentle pressure a quantity of milk into a long, narrow beaker glass. If the breast has not been nursed within an hour, there should be no difficulty in obtaining at least an ounce of milk in this way. With this quantity we can at once decide on the efficiency of a nurse. The milk should have a bluish-white tinge. Any trace of yellow or green when a test-tube of the milk is held in the light, is abnormal. Milk may be very abundant but of a dirty white tinge; some specimens separate almost instantly upon withdrawal into a yellowish oily layer on top and a serous liquid below. Any such abnormalities in the milk should cause the rejection of an applicant. If the breasts, history, and physical examination are satisfactory, and the quantity and physical characteristics of a nurse's milk are

good, we may recommend her without making a chemical examination of the milk. Such an examination is impracticable for the practitioner with the means at his disposal. Even if carried out, it may be unfair to the nurse. At the examining visit the proportion of proteids and fats may be below what it will adjust itself to in a day or two when the wet-nurse is rested and housed in her new home. More nutritious diet will greatly change the composition of the milk. There are, however, conditions which may require an examination of the milk at a subsequent period. In such a case the methods to be hereafter detailed may be resorted to.

### **The Mother has Milk, but it is Insufficient in Quantity!**

It often happens that a mother has a small supply of milk in one or both breasts. The question arises: Should we reject such a breast and seek a nurse with an abundance of milk, or place the baby wholly on the bottle? I very strongly advise all such mothers to feed the baby partly at the breast and partly on the bottle. This is good morally in that it gives the mother the satisfaction of nursing her offspring. Furthermore, infants thrive better on mixed feeding of the breast and bottle than on the bottle alone. The breast milk seems to aid in the assimilation of the cows' milk. I follow this plan even if there are only two good nursings a day in one or both breasts of the mother. Again, it is common experience that suckling a breast in which secretion is at first deficient will develop and stimulate the secretion of the breast.

### **Contraindications to Nursing the Infant.**

If the mother is tuberculous, demented, or epileptic, the baby should not nurse the breast. On the other hand, she may suffer from syphilis or skin eruptions, or may have a deficiency of milk, and still be allowed to nurse her infant. A wet-nurse, on the contrary, must be free from all constitutional taint to be fit to nurse an infant other than her own.

### **Placing the Baby at the Breast.**

The question is constantly raised: Should the newly born infant be placed at the breast during the first twenty-four hours? If, after the babe is born, it sleeps and awakens only when the diaper is to be changed, and then falls asleep again, it is obviously not necessary to place it at the breast. If, however, the infant cries, is uneasy, and refuses to be quieted, we may, six hours after delivery, place it at the mother's breast. There are then a few drops of colostrum in the breast, and the infant will be quieted with this. Should this



not appease the infant, which rarely happens, it may be given a teaspoonful of sugar-water every two hours. On the second day there will, as a rule, be more milk in the breasts, and on the third day the secretion is more active, and may even cause caking of the breast.

### Care of the Breast.

The care of the mother's breast directly after the birth of the child is important. If we begin correctly, much trouble will be avoided.

**Caking.**—The breasts are closely watched to prevent so-called caking. If the baby nurses and leaves a residual amount of milk in the breast, this milk should be pumped off with an ordinary breast-pump. The most satisfactory pump is one with a glass cylinder and a rubber bulb. Pumping the breast at first, when the milk is forming, will prevent caking and rapidly regulate the secretion to a normal amount. If caking occurs, the breast should be rubbed with a massage performed three times daily. The hands of the nurse should be carefully washed, and anointed with some sterilized oil. The breast is then grasped in the palms of both hands, one above and the other beneath the breast. The breast is gently subjected to firm pressure with a vermicular motion. This massage is kept up five or ten minutes.

**Sore Nipples.**—Ordinarily, if the nipple of the mother or nurse is kept dry and clean, it will not fissure. Fissures, however, occur even when much care has been taken to prevent them. In such a case the baby should not nurse the breast directly, but through a rubber shield, which protects the nipple. The fissure is treated once daily with a 10 per cent. solution of silver nitrate. If there is a discharge of pus from the fissures, or if the breast nipple has become a focus of suppuration which discharges, the breast should not be nursed by the baby, as by so doing the infant may contract infectious diarrhoea.

### Nursing the Infant.

**Nursing.**—The baby should nurse about twenty minutes and then fall asleep at the breast. The nipple is washed, as already stated, with a solution of boric acid before and after each nursing. The breast nipple is covered in the intervals of nursing with a small piece of absorbent gauze folded several times. In this way the nipple does not come in contact with the clothing. Any exuding milk is caught on the gauze, which is replaced by a clean piece when necessary. The infant, when nursing, should lie in the arms of the nurse. The nurse grasps her breast just behind the base of the nipple with the index and ring fingers. The thumb may be used to exert pressure on the breast, thus aiding the flow of milk. In this way the inf-



is prevented from drawing the nipple too far into the mouth. Moistening the breast with saliva or a few drops of milk is reprehensible. The infant will furnish all the moisture needed.

**Intervals of Nursing.**—Up to the second month an infant should be nursed more frequently than at a later period. At this time eight nursings in the twenty-four hours are not excessive. From the second to the sixth month, seven nursings in the twenty-four hours are sufficient, and after the sixth month six nursings are sufficient. The nursings should be so arranged that the nurse and baby may have a complete rest of five hours between 12 P. M. and 5 A. M. The mouth of the infant is not washed (see above).

**Signs of Efficient Nursing.**—An infant at the breast whose weight increases in the regular ratio, who sleeps between the nursings, and whose bowels are normal, is known to be thriving. It may here be proper to give the normal weight curve (Fig. 11).

**Increase of Weight.**—During the first two or three days following birth the infant decreases in weight. Usually this loss is from 150 to 200 grammes (5 to 6½ ounces), but it is sometimes greater. The passage of meconium and urine, the exhalations from the skin and lungs, and the small amount of nourishment taken, account for this loss. As the infant begins to nurse, the weight increases until the seventh day, when it will have regained its original weight. On the tenth day it weighs 100 grammes (3½ ounces) more than at birth (Budin). In some cases, if the infant is placed immediately after birth on an abundantly secreting breast, it will not lose any or but very little weight.

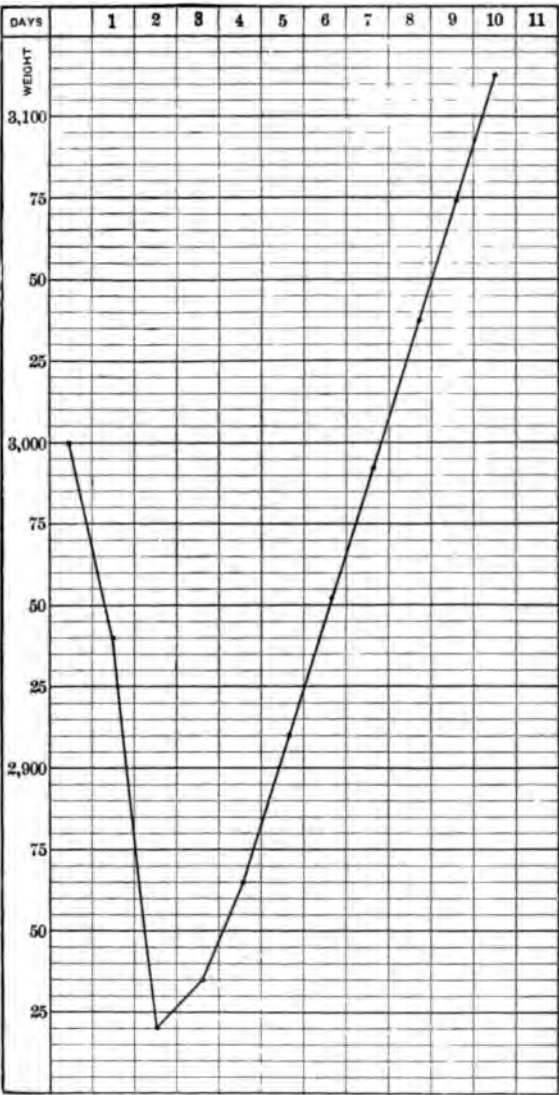
Cramerer gives a very instructive table which explains the loss of weight in the first two days after birth. It will be seen that the amount of nourishment is not sufficient to make up for the loss in weight.

	Nourishment taken, breast milk, in grammes.	Loss in grammes.	Change in weight, grammes.
First day . . . . .	30	190 { perspiration, 100 { urine, 50 { meconium, 40	— 160
Second day . . . . .	130	180 { perspiration, 80 { urine, 60 { meconium, 40	— 50
Third day . . . . .	240	230 { perspiration, 87 { urine, 140 { feces, 3	+ 10

In an investigation by Gundling it was noted that many infants ceased to lose after the second day, and an almost equal number on the third day. Boys lost more than girls, and the infants of multiparæ less than those of primiparæ. The average loss was 241 grammes. Most infants regained their original weight by the ninth day.

The tables on page 55 show the progressive increase in

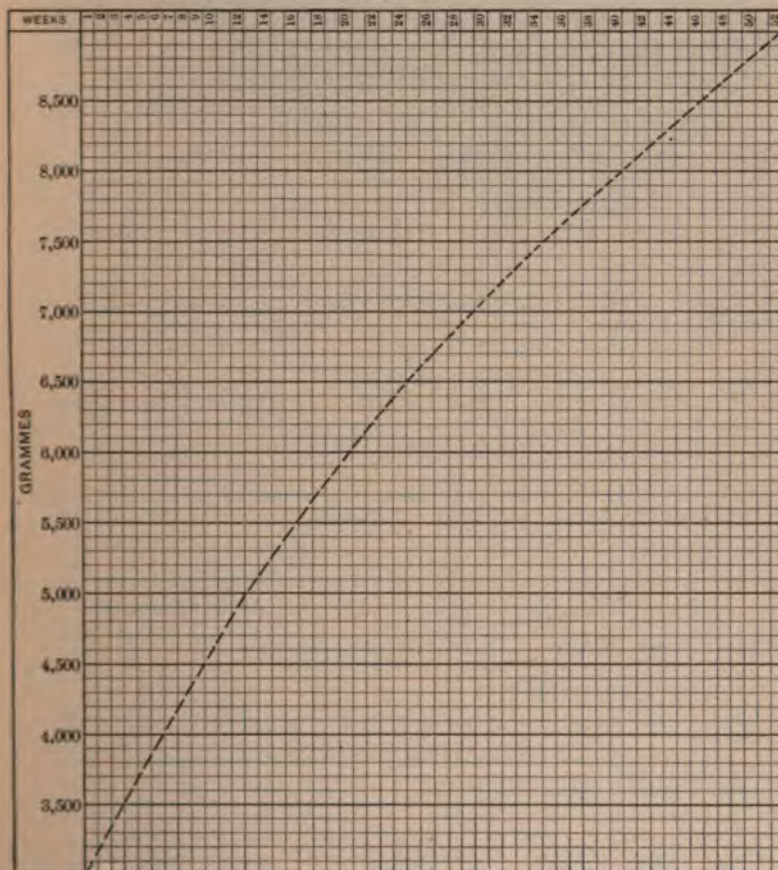
FIG. 11.



Normal curve of weight during the first ten days of life (Budln.)

After six months the infant has twice its initial weight, and end of twelve months weighs almost 20 pounds (9000 gramm

FIG. 12.



Normal weight curve of an infant during the first year. (Budín.)

*Normal Average Weight of Breast-fed Infants. (Camerer.)*

	Grammes.		Grammes.
Birth . . . . .	3450	End of 24th week . . . .	7130
End of 1st week . . . .	3400	" 28th " . . . .	7570
" 2d " . . . . .	3490	" 32d " . . . .	7990
" 4th " . . . . .	3890	" 36th " . . . .	8400
" 8th " . . . . .	4680	" 40th " . . . .	8580
" 12th " . . . . .	5410	" 44th " . . . .	9020
" 16th " . . . . .	6090	" 48th " . . . .	9300
" 20th " . . . . .	6650	" 52d " . . . .	9890

*Daily Increase in Weight in Grammes.*

Weeks.	Weeks.	Weeks.	Weeks.	Weeks.	Weeks.	Weeks.	Weeks.
<u>1-2</u>	<u>2-4</u>	<u>4-8</u>	<u>8-12</u>	<u>12-16</u>	<u>16-20</u>	<u>20-24</u>	<u>24-28</u>
3	29	28	26	24	20	17	15
		Weeks.	Weeks.	Weeks.	Weeks.		
		<u>28-32</u>	<u>32-36</u>	<u>36-40</u>	<u>40-52</u>		
		15	14	7	15		



For completeness, there may be added the measurements of length of the newborn infant, 49 to 50 cm. (Herz). Boys are the average longer than girls.

Herz gives the following measurements of the head of the born infant :

Circumference . . . . .	39.2 cm.
Sagittal diameter . . . . .	11.1 cm.
Large transverse diameter . . . . .	9.0 cm.

Weight from the first to the fifth year, by Camerer, in 1 (kilo = 2.2 pounds) :

	At birth.	First year.	Second year.	Third year.	Fourth year.	Fifth year.
Boys . . . .	3.4	9.9	12.8	14.9	16.7	18.0
Girls . . . .	3.2	9.2	14.9	13.2	15.0	16.0

American-born infants and children have the following weight and length of body :

			Length.	
			Boys.	Girls.
End of first year . . . . .			19½ inches.	19 inches.
“ second year . . . . .			27 “	26 “
“ third year . . . . .			33 “	33 “
			Weight.	
			Boys.	Girls.
End of first year . . . . .			20 pounds.	18-19 pounds.
Second year to fourth year . . . . .			28 “	26 “

The following tables by MacDonald are taken from studies of children above five years of age :

*Average Boys, American Parents.*

Limits of different ages.				Total number of children.	Average height.	Average sitting height.	Average weight.	Average circumference of head.
From—		To—						
Yrs.	Mos.	Yrs.	Mos.		Inches.	Inches.	Pounds.	Inches.
5	7	6	6	36	44.64	24.85	45.01	20.21
6	7	7	6	175	46.13	25.19	48.07	20.35
7	7	8	6	285	47.95	25.94	51.76	20.53
8	7	9	6	286	49.79	26.55	56.17	20.63
9	7	10	6	305	51.63	27.18	61.43	20.75
10	7	11	6	320	53.24	27.98	66.40	20.85
11	7	12	6	289	55.17	28.71	73.19	20.90
12	7	13	6	360	56.71	29.39	79.94	20.96
13	7	14	6	311	58.99	30.60	88.48	21.27
14	7	15	6	196	61.82	31.63	99.83	21.40
15	7	16	6	119	64.11	33.12	114.28	21.64

*Girls of American Parentage.*

Limits of different ages.				Total number of children.	Average height.	Average sitting height.	Average weight.	Average circumference of head.
From—		To—						
Yrs.	Mos.	Yrs.	Mos.		Inches.	Inches.	Pounds.	Inches.
5	4	6	6	64	44.47	24.36	43.52	19.88
5	5	6	11	37	43.97	23.87	42.90	20.20
6	5	7	6	375	45.09	24.69	45.74	19.94
7	7	8	6	587	47.43	25.49	49.33	20.13
8	7	9	6	669	49.21	26.19	53.47	20.29
9	7	10	6	737	51.22	27.00	58.53	20.45
10	7	11	6	721	53.15	27.81	63.98	20.55
11	7	12	6	673	55.81	29.06	72.83	20.78
12	7	13	6	706	58.05	30.21	82.42	20.97
13	7	14	6	627	60.25	31.43	92.96	21.18
14	7	15	6	490	61.64	32.23	100.69	21.29

**Movements of the Bowel.**—An infant's stool is normal if it is yellow in color, of the consistency of thick paste, and does not contain white flakes. The pasty, unformed stool is characteristic of the infant. If the feces are passed in round masses, the movement is abnormal. If a dejection presents white or greenish masses, contains mucus to an appreciable degree, is green when passed, or is partly pasty and partly fluid, it is abnormal. Any movement may turn green after exposure to the air for any length of time.

The number of movements passed daily varies. Some infants have regularly one normal movement daily; others, two or more. The author has met infants in good health who had at times six normal movements daily. Henoch has called attention to this fact.

**Signs of Insufficient Breast-feeding.**—A baby is not thriving on the breast if its weight remains stationary for even a short time. For this reason babies should be weighed at least twice a month. At the first discovery of stationary weight an infant should be weighed every three days, in order to see if it increases in weight. If the weight continues stationary, the milk should be examined. It may be deficient in quantity to such an extent as to no longer fully satisfy the baby. In that case the infant will be observed to nurse the breast for a long time; or it may nurse the breast a short time and then relinquish it and begin to cry; or it may cry in the intervals of nursing. All these are signs of insufficient food. In such cases, the breast is examined just before a regular nursing, in order to estimate the quantity of milk in the breast. The breast is also examined after nursing. In this way the physician can determine whether the quantity of milk secreted is sufficient. The movements of insufficiently fed infants are dry and constipated. The author has seen the character of the stools improve upon increasing the quantity of food, either from the breast or by supplementing the breast with the bottle. In some cases the baby cries and has colic, the movements are passed with a good deal of

flatus and are irregular in consistency and color. Here the quantity of the milk may be sufficient, but its quality is not up to required standard. The nurse's milk should be examined—excitement may have caused a change in it. Under the heading of Milk and Its Analysis, it will be shown that regimen and exercise can much to remedy deficient milk.

### Composition of Human Milk.

Human milk is so variable in composition that definite knowledge on many aspects of its chemistry is still lacking. The older analyses of human breast milk give the gross amount of proteids. Hoppe Seyler suggested that the casein should be determined apart from the total proteids. Analyses which deal with total proteids are not useful to the physician as those which deal with the casein apart from the proteids. The importance of this point will become apparent on comparing the milk of the human being with that of the cow.

König's analysis, as modified by White and Lladd, gives the following composition of human milk and cows' milk:

	Cow.	Human.
Caseinogen . . . . .	2.88	0.59
Whey proteids . . . . .	0.53	1.25
	3.41	1.84

The casein in cows' milk comprises five-sixths of the proteid in human milk, two-sixths of the total amount. We should bear this important fact in mind in reading the following tables compiled from Camerer and Söldner, showing the composition of human milk.

	Ether ext. fat.	Milk- sugar.	Proteids.
Colostrum . . . . .	5.0	4.5	3.5
Milk, fifth day . . . . .	2.3	6.7	1.6
Milk, ninth day . . . . .	3.4	6.7	1.4
Milk, first month . . . . .	2.6	7.3	1.1
Second and third months . . . . .	2.4 to 1.9	7.5	0.9

Backhaus gives the following table of average composition (in 100 parts) of human milk:

Water . . . . .	88.20
Proteids . . . . .	{ 0.75 casein. 1.00 albumin (whey proteids).
Fat . . . . .	3.50
Sugar . . . . .	6.20
Ash . . . . .	0.25

On comparing these figures with those of König, White, and Lladd, it will be seen that White and Lladd include all the proteids



exclusive of casein under the name of whey proteids. The whey proteids are principally lactalbumin and lactoglobulin.

Human milk is a bluish-white fluid, amphoteric in reaction. It is relatively more alkaline but absolutely less so than cows' milk. The specific gravity ranges from 1028 to 1034. It is lower in poorly nourished women. The essential difference between human and cows' milk lies in the casein. Human milk is not only poorer in casein than cows' milk, but the casein is less in proportionate combination with the remaining proteids, lactalbumin, etc., than in cows' milk. This in part explains the more flocculent nature of the casein coagulum in human milk apart from its alkalinity, which must also have an influence on coagulation, as noted in the chapter on Stomach Digestion. Human milk contains, in addition to lecithin and nucleon, more combined phosphorus than cows' milk in the nucleon (Siegfried).

The casein of human milk is derived from the protoplasm of the cells of the mammary gland. Fat is set free from the cells of the gland in which fat is formed. Iodine, arsenic, antimony, zinc, lead, mercury, and iron pass from the blood into the milk of the human breast. Iodine has caused iodism in the nursing infant when taken by the nurse in the form of the iodides (Koplik).

These facts are of practical interest to the physician. Very important is the fact that not only does the total of proteids and fats in the milk of various women vary to the extent of 1 per cent. or more, but also the milk of the nursing woman varies at different hours of the day, as may be seen in the following table by Schlichter :

Nurse A.	Casein.	Fat.	Proteids.	Sugar.
Morning . . . . .	1.10	0.80	1.69	7.11
Noon . . . . .	1.10	1.88	2.16	6.92
Night . . . . .		3.16	1.95	6.83
Nurse B.				
Morning . . . . .	0.55	3.77	1.19	5.37
Noon . . . . .	0.77	3.90	1.91	6.15
Night . . . . .	0.71	3.73	1.26	6.19
Nurse C.				
Morning . . . . .	0.55	3.61	0.19	6.18
Noon . . . . .	0.83	4.21	1.08	6.24
Night . . . . .	0.41	3.60	1.16	6.47

#### Can the Composition of Woman's Milk be Altered at Will?—

I think that this question is still *sub judice*. Although it is possible to improve the nurse's milk by increasing the general nutritive qualities of her diet, we cannot always cause increase or diminution in the casein or fat by giving certain articles of food. If a nurse has been on an insufficient diet, it should be increased in a general way. She should have a moderate allowance of meat, partake sparingly of tea, coffee, and beer, and have sufficient exercise. If the milk does not improve, the nurse should be replaced by another. If the

infant has been suckled by its mother, the deficiency if only one quantity should be supplied by mixed feeding.

**Influence of Foods on the Breast Milk.**—A diet rich in nitrogenous substances increases the quantity of the milk and the percentage of fats and proteids. A diet rich in fats may increase the fat of the milk. On the other hand, it is not always possible to increase by means of the diet the casein in milk poor in this constituent (König). Starvation lessens the quantity of the milk and the proportion of the casein to the other proteids, as does also a poor dietary (Decaisne). Enriching the diet improves the milk. Beer and malt liquors increase the quantity of milk and its fat constituent (König). In this connection, we should not lose sight of the fact that the milk of a good breast may be made unfit for the infant by placing the nurse on a diet to which she is unaccustomed. Women accustomed to a wholesome, moderate dietary will, if fed liberally with fats and carbohydrates, secrete a milk rich in fat and poor in proteids. This will at once disagree with the infant (König).

Menstruation has no appreciable effect on the milk. Pregnancy does not always change the milk perceptibly in the first months. This has been my experience, and it corresponds with that of Budin. Most authors assert that with the advent of pregnancy the milk changes into colostrum. The milk of a pregnant woman should be discontinued after the third month. Up to that time it is well to examine the milk and the baby in order to determine the fitness of the milk for the baby, and also its continued well-being on such milk.

The presence of drugs and aromatics in the milk has been mentioned. The author has seen the milk of women who had eaten asparagus disagree with infants. Excessive purging with pills containing aloes and belladonna may have a similar effect.

#### **Bacteria in Breast Milk.**

It is now known that bacteria exist in the breast milk of women in good health. The bacteria belong principally to the *Staphylococcus albus* class. *Staphylococcus pyogenes aureus* and some few streptococci have been found (Cohn and Neuman). On the other hand, staphylococci and streptococci are quite frequently found in the milk of women whose breasts are suppurating or are affected with rhagades and fissures. Escherich found bacteria in the breast milk of women suffering from puerperal sepsis. Pneumococci have been found in the milk of women suffering from pneumonia (Foa, Bordoni-Uffreduzzi).

#### **Methods of Analysis of Human Milk.**

In the section treating of the examination of the milk of the nurse or mother it was shown that with experience it is possible to decid



in a general way as to the quality of the milk without chemical analysis. Emergencies, however, arise which may necessitate more careful examination of the milk in order to clear up some disturbing symptom in the infant. After thriving for a few weeks the infant may, without apparent cause, cease to gain in weight, or the movements may be abnormal, or there may be colic. Under these conditions it is certainly an advantage to be able to determine the composition of the milk, since a chemist is not always at hand. Conrad, a physician in Berne, has devised some instruments which are easily manipulated and are within the reach of every physician. His article, published in 1880, is still unsurpassed in clearness of detail. The milk to be used in all analyses is that obtained in the mid-period of nursing.

**Specific Gravity.**—To ascertain the specific gravity, Conrad reduced the size of Quevenne's lactodensimeter so that it could be utilized for taking the specific gravity of small quantities of mother's milk (Fig. 14). The specific gravity is taken at 15° C. The scale runs from 1020 to 1050.

**Fat.**—Conrad estimated the fat by first calculating the cream layer. This he determined by means of a graduated glass cylinder devised by Bouchardat, Quevenne, and Chevalier. This cylinder he reduced in size. The method is so unreliable that it is merely mentioned in passing.

Of greater reliability is the

FIG. 13.



Conrad's lactobutyr-ometer.

FIG. 14.



Conrad's lactoden-simeter.



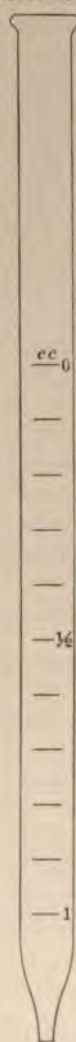
FIG. 15. Marchand tube, reduced in size by Conrad. The set consists of two of these tubes. Each tube analyzes 5 c.c. of milk (Fig. 13).

Five c.c. of milk are poured into the tube, and then 5 c.c. of ether. These are well shaken after a drop of officinal caustic soda solution has been added. Absolute alcohol is then added up to the

FIG. 16.



FIG. 17.



A mark. The whole is again shaken and placed in water at 35° to 40° C. for ten or fifteen minutes. The fat separates above, and is read off. A percentage table accompanies the instrument. This instrument is not accurate. There is a variation of from 0.2 to 0.5 per cent. or more. Two analyses are made at the same time for the sake of accuracy; hence the two tubes.

**Lewi's Method.**—More accurate than Conrad's is the method worked out in my clinic by Lewi. This is really an adaptation to mother's milk of the Babcock sulphuric acid method, as modified by Leffman and Beam.

The apparatus needed comprises a reduced Babcock bottle, a pipette for measuring the milk and acid, and a smaller 1 c.c. pipette accurately divided into cubic millimetres (see Figs. 15, 16, and 17).

**METHOD.**—Fill the pipette to the meniscus (this represents 2.92 c.c. of mother's milk), and introduce this carefully into the body of the bottle, so that the long, thin pipette comes down into the body of the bottle. The pipette is cleansed, and re-

Instruments employed in the estimation of fat in milk. Lewi's method.

filled to the meniscus with chemically pure sulphuric acid; the pipette is introduced as before. This precaution is taken in inserting the pipette so that at this stage no ebullition shall occur in the neck of the bottle, and so invalidate the result. Next, fill the 1 c.c. pipette up to the sixth marking with a mixture of equal parts of fusil oil and concentrated hydrochloric acid; add this to the milk and sulphuric acid, and fill the bottle with equal parts of sulphuric acid and water. The bottle is placed in an aluminum receiver and adjusted to the centrifuge. The specimens are revolved one and a half to two minutes, and the reading is then taken. With the new high-gear machine of Richards & Co. fifteen revolutions of the handle per minute suffice, each turn of the handle corresponding to one hundred and thirty revolutions of the bottle.

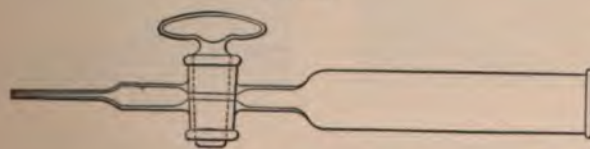
This method, if carefully carried out, gives very little error, and is practically equal to the Soxhlet quantitative fat estimation. It can be applied to cows' as well as to human milk.

The following table shows the error in the various methods as compared with accurate chemical determination:

	Soxhlet (chemical).	Reduced centrifuge.	Marchand.	Feser.
Specimen I. . . .	4.4 per cent.	4.4 per cent.	3.48 per cent.	5.00 per cent.
" II. . . .	2.4 "	2.3 "	2.56 "	2.37 "
" III. . . .	1.1 "	1.1 "	1.44 "	1.25 "
" IV. . . .	3.9 "	3.8 "	3.17 "	3.25 "
" V. . . .	4.6 "	4.7 "	2.35 "	3.80 "
" VI. . . .	2.3 "	2.3 "	3.99 "	2.20 "
" VII. . . .	4.4 "	4.2 "	3.68 "	4.20 "
" VIII. . . .	4.7 "	4.6 "		3.60 "

**The Proteids.**—To possess clinical value in the determination of the proteids, a method must differentiate between the amount of casein and that of the other proteids, such as lactalbumin and lactoglobulin. This is possible only by careful and exhaustive quanti-

FIG. 18.



Milk burette of Woodward.

tative chemical analyses. The methods at our disposal which are practicable in the physician's office determine only the gross proteids. The gross proteids may be normal in amount, and the casein or



caseinogen be deficient. Such milk would not be nutritious. was demonstrated years ago in sick and starving women (Decai

The following is the method of Woodward for determining total proteids: Two "milk burettes" (Fig. 18), each containing 5 c.c. of milk, are allowed to stand overnight in a warm place (100° F., 38° C.). They are then cooled. The milk is drawn into two Esbach's tubes, and 10 c.c. of the Esbach solution added. The tubes are then shaken, put into a centrifuge, and rotated until the reading is constant. This method was perfected in the Peck Laboratory, Philadelphia. The author has applied it in a number of cases, and found it satisfactory.

### Microscopical Examination of Human Milk.

On placing a drop of human milk under the microscope, highly refracting spherical bodies are seen. They vary from 0.0024 to 0.0046  $\mu$  in diameter (Wall) (Plate IV.). Up to the eighth day after the birth of the child the breast milk may, in addition, contain the so-called colostrum corpuscles. These are spherical bodies four or five times larger than milk bodies. They consist of granular masses of fat. They are formed in the later months of pregnancy and disappear in the course of a week or two after birth. Colostrum corpuscles may appear in the milk at any time during the nursing period, and are then a sign of intercurrent pregnancy or disease. If abundant, they make the milk unfit for infant consumption.

### ARTIFICIAL FEEDING OF INFANTS.

An attempt has been made, especially in France, to rear infants on ass's milk, which has a composition identical with that of a woman's milk. The experiment has, however, failed. To-day cow's milk is universally utilized in substitute feeding. Before cow's milk can be made suitable for this purpose it must be modified, that is to say, the relative proportions of the casein, fat, and water must be changed.

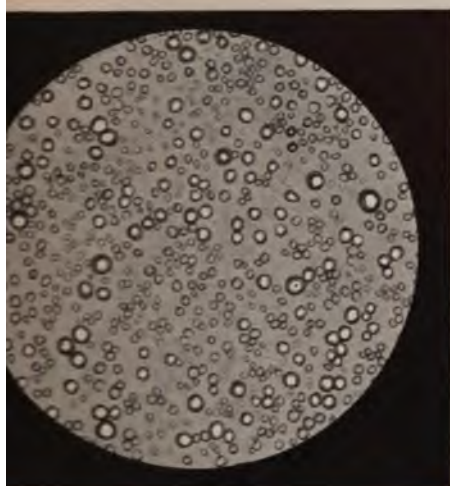
### Composition of Cows' Milk.

König gives the following as the average composition of cow's milk per 100 parts: water, 87.1; casein, 3.02; albumin, 0.5; fat, 3.69; sugar, 4.88. Cows' milk has a specific gravity from 1028 to 1034. It is amphoteric in reaction, but is relatively more acid than human milk. Fresh cows' milk does not coagulate on boiling, but heat causes a skim of casein and lime salts to appear.

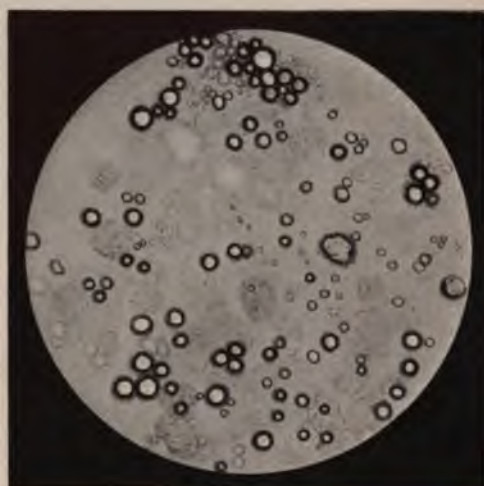


# PLATE IV.

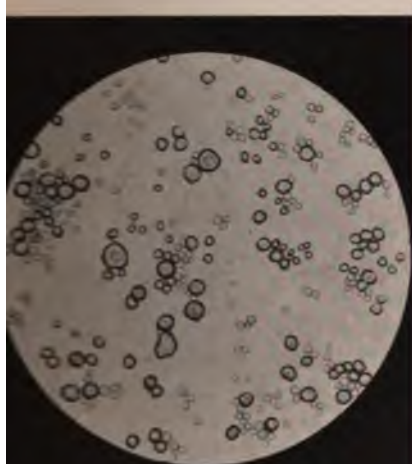
1.



2.



3.



4.



## Microscopical Appearances of Woman's Milk. (After Fleischman.)

1. Normal milk, showing the preponderance of medium-sized fat-globules.
2. Colostrum of later pregnancy.
3. Poor milk. Preponderance of large fat-globules and a paucity of fat.
4. Poor milk, a paucity of fat and an almost granular state of the fat-globules.

quantity of milk necessary to maintain nutrition is greater than the case of the breast-fed infant. There is always the danger of overfeeding an infant on the bottle. The increase in weight is so regular as in the breast-fed infant, as is shown in the following table :

Camerer	Weeks	1—2	2—4	4—8	8—12	12—16	16—20	20—24	
	Increase	4	21	21	22	22	25	22	
	Weeks				24—28	28—32	32—36	36—40	40—44
	Increase				13	16	16	9	
Koplik	Months	1	2	3	4	5	6	7	8
	Weight	3810	4430	5090	5800	6550	7180	7650	8140
Koplik	Months	1	2	3	4	5	6	7	8
	Increase		3735	5068	5285	5518	7688	7223	8680

In the above tables there are shown in grammes not only irregularity in the daily increase, but also the irregularity in the weight. My own cases were examined with a view to determine what an artificially fed baby weighs if it is thriving. The figures correspond closely to those given by Camerer.

The following table shows in grammes the daily increase of weight of the breast-fed and the bottle-fed infant :

Months.	Ahlfeld (breast).	Camerer (bottle).	Koplik (bottle).
1 . . . . .	31	21	32.0
2 . . . . .	26	22	17.4
3 . . . . .	24	22	23.6
4 . . . . .	21	25	18.0
5 . . . . .	18	22	14.2
6 . . . . .	15	13	11.8
7 . . . . .	15	16	15.6
8 . . . . .	16	16	15.1
9 . . . . .	9	9	—

### The Modification of Cows' Milk for Infant Feeding.

In order to make clear to the student the rationale of our present methods of artificial feeding, it will be of advantage to study the development of infant feeding with cows' milk. The casein of cow's milk has from the outset been a point of attack. The older method consisted of two or three dilutions of cows' milk. In the first month the milk was diluted 1 in 3 ; in the second month, 1 in 4 ; in the third month, 2 in 3. These simple methods continued in vogue until Biedert in Germany and Meigs in the United States attempted to proportion the casein, fat, and sugar as to make the mixture approach the composition of mother's milk.

Biedert called his food "cream mixture." It was made in the same general way as Meigs's mixture. These men perfected their formulas independently of each other, and, strange to say, insisted on the same fact—the low percentage of proteids in mother's milk.



Meigs claimed that the proteids ranged from 1.2 to 1.5 per cent. Biedert's mixture was constructed to contain—proteids, 1 per cent.; fat, 2 to 2.5 per cent.; sugar, 4 per cent. Meigs's mixture contained 3.5 per cent. of fat and 6 per cent. of sugar.

Biedert took the cream (0.25 litre) from milk (1.5 litres) that had stood one hour. This contained 10 per cent. of fat. With this he constructed the following formulas :

No. of mixt- ure.	Cream (10 per cent.) Litre.	Water. Litre.	Milk-sugar Gramme.	Milk. Litre.	Casein. Fat. Sugar.		
					Per cent.	Per cent.	Per cent.
I.	1	1	18	—	(= 1	2.5	5.)
II.	1	1	18	$\frac{1}{8}$	(= 1.4	2.6	5.)
III.	1	1	18	$\frac{1}{4}$	(= 1.5	2.6	5.)
IV.	1	1	18	$\frac{1}{2}$	(= 1.8	2.8	5.)
V.	1	1	18	$\frac{3}{4}$	(= 2.1	3	5.)
VI.	1	1	12	1	(= 2.3	2.4	5.)

If we compare these with Meigs's mixture, we find that Meigs constructed only one formula, which contained 1.2 per cent. of proteids, 3.5 per cent. of fat, and 6 per cent. of sugar. His method was identical with that described above. He used top cream, and mixed with it a certain quantity of milk and water (10 c.c. of cream, 5 c.c. of milk, 10 c.c. of lime-water, 15 c.c. of water, containing 2.2 grammes of milk-sugar).

In view of the subsequent trend of infant feeding, these two methods are of great interest. The method of Escherich has also been mentioned. It is based on an attempt to calculate by rough dilutions of milk the amount of albumin necessary daily for the maintenance of nutrition. There are two methods which are practically identical—the Heubner-Hoffman and the Soxhlet, in each of which an endeavor is made to obtain a mixture whose chemical equivalents will equal the nutritive calories in mother's milk. In both methods the milk is diluted with an equal part of water. Heubner uses as a diluent a 6 per cent. sugar of milk solution; Soxhlet, a 12 per cent. sugar of milk solution. The addition of sugar of milk is intended to supply the deficiency in fats. Soxhlet has shown that in the economy, sugar of milk has an equivalent caloric energy equal to that of the fat deficit. I have used the mixtures for years in a public laboratory and in private practice, and have found that they possess the following disadvantages: Infants from the first to the third month suffer from the diminution of fat and the large amount (1.8 to 2 per cent.) of proteids. The mixtures are suitable only for older children. With our present means we can construct mixtures more suitable to the needs of infants. This brings us to the consideration of the methods of infant feeding elaborated by Rotch. Rotch contended that all infants could not be fed on one mixture; that taking the compo-



sition of mother's milk as a working basis, each infant should be constructed for its use a formula which within certain limits would be most suitable to its needs. Rotch therefore separates milk from the cream by means of a separator. Working skimmed milk and cream containing 16 per cent. or 20 per cent. of fat and a solution of milk-sugar, the constituents of the milk are rearranged. By this method an infant can be fed on a mixture containing 0.5 per cent. of proteids, 3 per cent. of fat, and 6 per cent. of sugar; or 1.5 per cent. of proteids, 2.5 per cent. of fat, 6 per cent. of sugar; or any percentage of proteids, fat, and sugar that we may wish to give. He also contended that a baby would not thrive on 1.2 per cent. of proteids, might do so on 1.5 per cent. The proportion of fat might be reduced or increased as needed in the individual case. In other words, the physician should construct his percentage formula in feeding the infant just as he prescribes a certain strength of a drug. To obtain these percentages a laboratory is needed, and to-day laboratories for supplying mixtures to be used in the percentage feeding of infants are to be found in all large cities. Though this method of reconstructing milk is far the most rational yet proposed, it has certain inherent defects which Rotch and his pupils are trying to overcome. These defects are much the same as those of the older methods:

1. By simply rearranging the proteids, fat, and sugar we do not change the proportionate relationship which the casein or caseinogen bears to the lactalbumin and other proteids of the milk.

2. With the exception of a few limited facts and formulas, we have no data which with our present knowledge will enable us to know every case when to increase or diminish the proteids and also the fat.

3. It is contended by Starr, Monti, and others, though denied by Rotch, that the process of centrifuging impairs the original efficiency of the fat emulsion in the milk, and therefore the influence of this fat on the coagulation of the casein.

We cannot enter here into a consideration of the merits of the last objection. It is enough to grant at once that the work of Rotch has been of far-reaching importance in infant feeding. It has been a great educator to physicians and a simplifier of former confusion. We can now obtain good, fresh milk, and it may be modified according to any formula.

**The Whey Method of Dilution and Modification.**—The principal obstacle to the universal success of the original Rotch method, as stated in (1), that the proportion of the casein or caseinogen to the remaining proteids of the cows' milk (cows' milk, five-sixths caseinogen and one-sixth lactalbumin and lactoglobulin, as compared with mothers' milk, two-sixths caseinogen and four-sixths lactalbumin and lactoglobulin) remains the same. The most rational method of overcoming this obstacle is to dilute with whey instead of water.

to use a cream which is highly concentrated for the fat proportions, and to use skimmed milk to obtain the caseinogen.

This method was first proposed by Vigier, and elaborated by Monti, of Vienna, in 1897. It really produces a species of humanized milk. Monti took a quart of milk, added rennet to it, and heated it at 35° C. (95° F.) for twenty to thirty minutes. He then filtered off the curdled casein and pasteurized the whey. The pasteurized whey contains about 1 per cent. of proteids (lactalbumin and lactoglobulin). He then mixed the whey with equal parts of milk or with 1 part of whey and 2 parts of milk. He thus obtained 1.2 to 1.6 parts of casein with 1 per cent. of dissolved proteids in the whey. White and Ladd, pupils of Rotch, have still further reduced the casein, so that with concentrated cream, skimmed milk, and whey they obtain mixtures in which the caseinogen or casein bears the same proportionate relationship to the lactalbumin and lactoglobulin as it does in human milk. Thus, with a total proteid percentage of 1.25, two-thirds is whey proteids (lactalbumin, etc.) and one-third casein and caseinogen. The following table shows a few of the combinations of caseinogen and lactalbumin obtainable at the laboratories (Rotch):

Fat. Per cent.	Caseinogen. Per cent.	Lactalbumin. Per cent.	Sugar. Per cent.
1.00	0.25	0.25	4 to 7
1.50	0.25	0.75	4 to 7
2.00	0.50	0.75	4 to 7
2.50	0.50	0.75	4 to 7
3.00 or 3.50	0.50	0.75	4 to 7

We have thus arrived at a method by which mother's milk can be exactly duplicated. Its success in practice must be left for future study.

Having traced the development of the complex problem of infant feeding, we shall now consider its practical application.

### **Bacteria in Cows' Milk; Pasteurization; Sterilization.**

By insisting on strict cleanliness of the cow's udder, the hands of the operator, and the utensil in which the milk is collected, it is possible to obtain milk free from bacteria. Commercially, however, this is manifestly impracticable. Even milk collected with great care contains bacteria. If these bacteria number 9000 to the cubic centimetre, they will under favorable conditions increase, so that in twenty-four hours at ordinary temperatures they will number 5,600,000 (Miquel). The bacteria chiefly found in milk are the *Bacterium lactis aërogenes*, the *Bacillus mesentericus vulgatus* (potato bacillus), and the *Bacillus subtilis*. The milk may contain streptococci (*Escherich*) from the udder of the animal. It may contain any of the various pathogenic bacteria—pneumococci, typhoid bacillus, diphtheria bacillus, the germs of scarlet fever, measles, or the *Bacillus tuber-*



culosis. Milk is an excellent culture-medium for the germs of infectious diseases.

**Milk Acidity.**—If milk is not cooled immediately after withdrawal and kept cool, it soon shows a marked increase in acidity. This is due to the growth of the *Bacterium lactis aerogenes*. This micro-organism not only turns milk "sour," but by the production of toxins causes disturbances of the stomach and gut of nursing infant. Without entering into details foreign to this work it is sufficient to state that milk for infant feeding should be obtained from a herd of healthy cows. Mixed milk is to be preferred to that from one cow. The milk should be carefully collected into utensils which have been sterilized. Milk should reach the consumer as soon as possible after milking (within twenty-four hours at most). After being modified, the milk is divided into portions, each of which is sufficient for a nursing. It is then heated, in order that it may be kept unchanged for at least twenty-four hours.

**Pasteurization.**—This process was perfected by Pasteur. The milk is subjected to a temperature of 65° C. (149° F.) for a variable length of time, generally half an hour, and then rapidly cooled to 20° C. (68° F.). An excellent apparatus for this purpose, called the Freeman pasteurizer, has been devised by Freeman (Figs. 19, 20).

FIG. 19.

FIG. 20.



Freeman Pasteurizer.

If properly carried out, pasteurization destroys all pathogenic germs which may be present in the milk. It also destroys most of the *Bacterium lactis*, but will not destroy any sporulated bacteria such as the *Bacillus mesentericus vulgatus*.

**Sterilization** is the process of heating milk to 212° F. (100° C.). This may be done by means of the Arnold steam sterilizer (Fig. 21) or by simply placing the milk in properly corked bottles in boiling water. As a rule, the milk is heated for twenty minutes, when it is considered sterilized. The milk should then be rapidly cooled. The process of cooling prevents the separation of the fat into large globules and the breaking up of the natural emulsion of the milk.



Sterilization, as practised with the ordinary sterilizer, will not render milk sterile. It will not destroy any sporulated bacteria, but will destroy the *Bacterium lactis* and all pathogenic germs.

As has been stated, milk should be of good quality and contain a minimum of bacteria before being submitted to any heating process, for neither pasteurization nor sterilization will make milk which is decomposed fit for use. Both procedures are simply means which enable us to keep milk unchanged for a longer or shorter time.

The chief objections to sterilization of milk are that the casein is made less soluble, that the fat is separated in the form of butter, and that the milk has a "boiled" flavor. According to some authorities, sterilized milk which has been subjected to a temperature of 212° F. (100° C.) is less digestible than pasteurized milk that has been heated to 149° F. (65° C.).

FIG. 21.



Arnold Steam Sterilizer.

	Nitrogen taken in milk. Grammes.	Nitrogen remaining in feces. Per cent.
First infant—		
Pasteurized milk . . . . .	10.9209	4.6
Sterilized milk . . . . .	13.7449	4.9
Raw milk . . . . .	5.3914	3.4
Second infant—		
Boiled milk . . . . .	32.643	4.5
Sterilized milk . . . . .	30.969	4.3

The table given above shows the comparative digestibility of raw, pasteurized, and sterilized milk (Koplik), as indicated by the percentage of nitrogen remaining in the feces of the infant. These experiments were performed by feeding the same infant with raw and heated milk. The results showed that, although the differences are slight, they are in favor of milk subjected to little or no heat. Doane and Price have confirmed these results by experiments on the calf.

The part played by sterilized milk in the causing of scurvy has been elsewhere considered. Without entering deeply into a question which remains to-day rather unsettled, it may be said that the majority of experienced men believe pasteurization to be the less objectionable of the two methods. During the winter and spring months pasteurization answers all purposes. During the summer, if milk is transported not too great a distance and can be obtained within twelve hours of milking, pasteurization with subsequent packing in ice will keep it unchanged for twenty-four hours. Among the poor of large cities it is not always possible to obtain

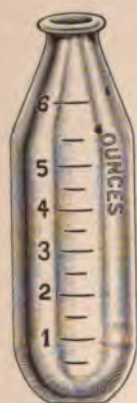
the milk early. Even if it is obtained fresh, the ignorance and carelessness of these people is so great that I have found it safe to sterilize the milk during the heated term. In this way gastro-enteric disturbances are avoided. On the other hand, milk obtained from reliable sources and carefully pasteurized within a few hours of the milking can be kept on ice unchanged for twenty-four hours in the summer. During the heated term we should never lose sight of the fact that pasteurized milk, if not kept on ice, increases acidity and causes gastro-enteric disturbances.

### Raw Milk in Infant Feeding.

Some physicians, in order to avoid the objections to pasteurization and sterilization, have proposed the use of raw milk in infant feeding. That is to say, the milk, after modification, is not heated but is kept on ice until needed. I have not been able to reach a conclusion on this point. Any unheated animal food is distasteful, no matter how great the care taken in its handling. It is noteworthy that infants who have suffered from scurvy will thrive on raw milk, while other infants will not. Adults with whom raw milk will not agree, tolerate well-heated or boiled milk. I have occasionally seen infants in whom raw milk, no matter how fresh, caused acid movements and green curds in the stools. Exceptional cases are met with in which pasteurized milk causes movements which are alternately fluid and formed in character. In these cases sterilized milk is well borne.

### The Nursing Bottle.

The best form of bottle is one that has very little neck, a wide mouth, and not much shoulder at the neck, so that it may be easily cleaned. These requirements are met by the Freeman bottle (Fig. 22). When filled, the bottles are corked with non-absorbent cotton. They are corked loosely, so that the steam may escape. If the cotton is jammed tightly into the bottle, the cork will blow out in heating. After the nursing, the bottles are filled with a saturated solution of washing soda and allowed to stand a few hours. They are then washed both externally and internally, and drained dry. Any residue of milk remaining after nursing should not be utilized for another nursing.



Nursing bottle  
of the Freeman  
model.

Nipples should be boiled once daily for ten minutes, and washed with hot water after each nursing.

Before feeding, the bottle of milk is warmed to 105° F. (40.5° C.), so that the milk may not chill the stomach of the infant and thereby suspend digestive processes.



**Quantity to be Fed to the Infant.**

The quantity of milk to be given at each feeding has been variously estimated. It is evident that the capacity of the stomach alone is a crude and most unscientific standard, nor is the age of the infant in itself a guide. More rational is the method which takes into consideration not only these data, but also the normal quantity of milk taken by a breast-fed infant daily and at each feeding. If, in addition, we can calculate the amount of albumin or proteids and fat necessary per kilogramme of the body weight to maintain nutrition, we shall have the most satisfactory method of determining the quantity of milk to be taken daily by the infant and the quantity to be given at each feeding. This method was first proposed by Biedert, and applied by him and Escherich. It was found that the nutrition of the infant could not be maintained by an amount of proteids of cows' milk equal to that taken in the breast milk. In other words, the proteid equivalent could be obtained, but the other constituents (fat, etc.) were at fault as well as the daily quantity of food. Escherich's own figures give a greater daily percentage of proteids than is found in mother's milk. On the other hand, the recent modifications of milk proposed by Backhaus, Rotch, Monti (whey mixture), White and Ladd give promise that the ideas of Biedert and Escherich may be carried out.

**Amount of Breast Milk Consumed by the Infant in Twenty-four Hours.**

Camerer has collated and analyzed the results obtained by Ahlfeld, E. Pfeiffer, Weigelin, and Hühner, as to the quantity of breast milk taken daily by an infant. The method of ascertaining the figures recorded by these authors was to weigh the infant before and after nursing. Camerer gives the following table:

Day.								
1st.	2d.	3d.	4th.	5th.	6th.	7th.	10th.	14th.
30	130	240	290	330	365	400	450	500

Amount of Milk Taken.					
	Middle 2d week.	4th week.	7th week.	10th week.	20th week.
Minimum . . .	210	380	520	600	700
Medium . . . .	440	580	770	800	900
Maximum . . .	540	810	1040	1170	1150

It is noteworthy that on the first day of life the infant observed by Camerer nursed three times, and seven times in each twenty-four hours from the second to the fourteenth day. Each nursing occupied a mean of about twenty to twenty-five minutes. These data are of value in prescribing for the artificial feeding of infants.



### Number of Nursings Daily, with the Necessary Quantity Each Feeding, in Artificially Fed Infants.

If we now attempt to apply the knowledge acquired in the study of the feeding of the breast-fed infant to the artificially fed infant we meet with the following obstacles: Cows' milk taken in the same quantities is not so completely used up by the economy as breast milk. There is much more waste, as has been shown by Knoepfmacher and Camerer. This waste is chiefly caused by the infant's failure to consume the casein and fat. The stools also of the bottle-fed infant are more numerous and of greater total bulk than is the case with the breast-fed infant. Knoepfmacher has shown that the waste in the gut of phosphorus in cows' milk is sixteen times as great as the waste of that element in breast milk. In view of this lack of definite knowledge on these points, the quantities of modified cows' milk given at each feeding are still determined by experience alone. We have, it is true, been aided in this work by the study of the absolute quantity of proteids and fats necessary to the maintenance of nutrition.

*Table showing the Number of Feedings and Quantities of Modified Milk to be given to Artificially Fed Infants.*

Age.	Number of feedings daily.	Quantity at each feeding.		Total to be given in 24 hours.	
		C.c.	Oz.	C.c.	Oz.
First day . . . . .	3	10		30	1
Second day . . . . .	8	20		160	5½
Third day . . . . .	8	30	1	240	8
Fourth day . . . . .	8	40		320	10¾
Seventh day . . . . .	8	50		400	13½
Second week . . . . .	8	60	2	480	16
Fourth week to first month . .	8	60	2	480	16
Two months . . . . .	7 or 8	90	3	720-630	21-20
Three months . . . . .	7	120	4	840	28
Four months . . . . .	7	150	5	1050	35
Five months . . . . .	6 or 7	180	6	1080-1260	36-40
Six months . . . . .	6	210	7	1260	42
Seven and eight months . . .	6	240	8	1440	48
Nine months . . . . .	6	250	8½	1500	50

The increase in the amount of milk from the seventh to the ninth month is not so apparent, since at this period we, as a rule, begin to feed cereals in addition to the milk.

### Composition of the Food.

With the healthy infant there is little need of frequent change of formulas. There are certain rules which must be strictly adhered to in order that the infant be not upset at the beginning:

The total amount of proteids in the cows' milk mixtures must be very low for the newly born infant, certainly not exceeding 0.5 per cent. during the first week. After this time the proteids are increased to 1 per cent., and kept at this point until the third month, when they are increased to about 1.5 per cent., and kept there until the ninth month. In vigorous infants of heavy weight we may increase the proteids at the sixth month to 2 per cent., but I have never found it necessary to go beyond this limit.

**Fats.**—The fats in the first few days after birth should be low in amount (2–2.5 per cent.). After the second week to the third month I have always given about 3 to 3.5 per cent. of fat. The reason for this is that during this period we find that the infant does not increase in weight as it should unless the fats are high. After the third month the fats may be reduced to 2 or 2.5 per cent. with most infants. This proportion is continued until the ninth month without change. Unless there is some special indication, I would construct the following percentage formulæ for *normal* infants :

Age.	Proteids.	Fats.	Sugar.
One to seven days . . . . .	0.50	2.00	6.00
One week to three months . . . . .	1.00	3.50	6.00
Three months to six months . . . . .	1.20	2.50	6.00
	or 1.50	2.50	6.00
Six months to nine months . . . . .	1.70	2.50	6.00
	or 2.00	2.50	6.00

### Household Modifications of Milk for Infant Feeding.

The accuracy of the laboratory cannot be attained in home modification of milk. No matter how carefully the milk is put together, there are details which are impracticable at home. In the laboratory the milk-supply is controlled; the milk has a definite composition; the modification is more accurately carried out. On the other hand, many infants do quite well on household modifications, which are crude as compared with those of the laboratory. For sick children and in convalescence it is of great value to obtain a modification whose composition is definite and accurate. It can thus be known what mixture will agree with the patient. On the recovery of the patient we often find that a mixture approximating that of the laboratory can be made up at home, and that the infant will thrive on it. Laboratories are not always accessible. We should, therefore, have at our command methods which will replace those of the laboratory if only in a rough way. There have been devised many methods of so-called home modification. They have the merits and faults of mathematical formulas—they are rarely at



our finger-tips. I have not had an extensive experience in the use of these formulas. There are two very practical and easily remembered methods of home modification. One is that of Chapin. Chapin found that in milk which is put up in bottles the cream sets so that its composition is the same within certain limits. In city milk, the first 9 to 11 ounces of cream from the top of the bottle will contain 12 per cent. of fat. If 16 ounces are taken from the top of the bottle, the cream will contain about 8 per cent. of fat.

Age.	Composition.			Top cream.		Amount of food daily	
	Proteids.	Fat.	Sugar.				
1-7 days . . . .	0.50	2.0	6.0	2 oz.	diluted 6 times.	360 c.c.	12 oz.
1 week to 3d month . . . .	1.00	3.5	6.0	7 "	" 4 "	840.	28 "
3-6 months . . . .	1.12	2.6	6.0	14 "	" 3 "	1200.	42 "
6-9 months . . . .	$\left\{ \begin{array}{l} 1.76 \\ \text{to} \\ 2.00 \end{array} \right\}$	2.00	6.0	23 "	" 2 "	1400.	46 "

(Two bottles of milk required in order to get requisite amount of cream.)

The percentages given above are not absolutely correct. They will vary with the proportion of cream in the milk. They are as correct as any home modifications. We can start infants on these percentages and increase or diminish the fat or proteids if the infant does not thrive. In the table just given it is assumed that cows' milk has the composition of proteids 3.5 per cent., fats 3.6 to 4 per cent., and sugar 4 per cent.

The other method of which I have made use requires cream containing 16 per cent. of fat. With ordinary milk and a 6 per cent. solution of sugar of milk, assuming the composition of cows' milk as above, we first calculate the proteid composition of the mixture, diluting with the 6 per cent. sugar of milk solution. This gives us, if we dilute the milk, a mixture having the absolute proteids and a certain percentage of fat and sugar. The sugar is ignored. We then add to this the requisite amount of the 16 per cent. cream to bring the fats up to the required strength, allowing for the fat already in the dilution. This is simply a matter of calculation, and can be worked out by anyone if the required materials are at hand. In cities the laboratories will furnish the 16 per cent. cream. In other places a gravity or top cream containing 12 per cent. of fat can be utilized instead. The following table is constructed with 16 per cent. cream, ordinary milk, and 6 per cent. solution of sugar of milk.

The practitioner will find that the above milk modifications can be utilized in most normal cases.

All modifications of cows' milk must be alkalized with lime-water. This is the most practical and accessible alkali. A teaspoonful of lime-water is allowed to 8 ounces of milk.



*König's Analysis—Average Cows' Milk—87.1 Water, 3.5 Proteids, 3.6 Fat, 4 Sugar.*

Age	Modification.			Daily total quantity needed.	Constituents, 16 per cent. cream, milk, 6 per cent. solution sugar of milk.
	Proteids.	Fat.	Sugar.		
1-7 days . . . . .	0.5	2.0	6.0	Up to 350 c.c. 12 ounces.	<div style="text-align: right;">C.c.</div> { Cream (16 per ct.) 34.00 { Milk (whole) . . . 16.00 { Water(sugar 6%) 300.00 { Cream . . . . . 140.00
1 week to 3 months . . . . .	1.0	3.5	6.0	Up to 840 c.c. 28 ounces.	{ Milk . . . . . 140.00 { Sugar solution . 560.00 { Cream . . . . . 120.00
3-6 months . . . . .	1.2	2.6	6.0	Up to 1200 c.c. 40 ounces.	{ Milk . . . . . 280.00 { Sugar solution . 800.00 { Cream . . . . . 50.00
6-9 months . . . . .	1.7	2.0	6.0	Up to 1400 c.c. 46 ounces.	{ Milk . . . . . 650.00 { Sugar solution . 700.00

### Infant Foods.

There is no infant food, except those modifications of cows' milk described elsewhere, which can be utilized as a substitute for the breast with any success. In the case of older infants and young children the infant foods of a certain type may be utilized as adjuncts to the baby's dietary. This is done much as we would utilize any cereal.

Infant foods are divided first into those which are cereals in a pure state or subjected to heat, and thus dextrinized. To this class belong Imperial granum and Ridge's food. The other large class contains (a) milk mixed with cereals or (b) with forms of malt. In (a) are the foods of the Nestle food class, which contains evaporated and dried milk and cereal. In (b) are all the malted milk foods, such as Horlick's, malted milk, etc, Liebig's infant food in dry state.

If we study all these foods, including the condensed milk, we shall see that they show a deficiency of fat and an excess of carbohydrates. They should not therefore be used exclusively for a long time. Condensed milk also contains so much sugar that it causes acid dyspepsia. There are preparations of condensed milk made up without sugar. These are likely to decompose, and even when fresh have the disadvantages of all the artificial foods. A number of cases of scurvy resulting from the prolonged exclusive use of condensed milk and infant foods have been reported.

On the other hand, I have utilized the infant foods which do not contain any milk as adjuncts to milk modified or whole. In the treatment of enteritis, both of the acute and the subacute type, it is essential to give temporarily some food which does not contain milk in any form. With some form of cereal food such cases are very successfully tided over the period of subacute enteric catarrh. The

*Foods for Infants.*

Name.	Water.	Fat.	Protein.	Carbohydrates.		Fibre.	Ash.
				Soluble.	Insoluble.		
Carnrick's soluble food . . . . .	3.12	6.26	16.32	56.62	14.44	0.22	3.02
Horlick's food . . . . .	3.64	2.01	11.28	63.14	17.28	0.73	1.92
Horlick's malted milk . . . . .	2.87	7.81	16.61	59.43	10.95	0.52	1.81
Hubbell's prepared wheat . . . . .	5.93	1.19	14.81	16.16	60.86	0.31	0.44
Imperial granum . . . . .	10.57	1.32	19.37	15.42	51.88	0.31	1.13
Just's dietetic food . . . . .	4.83	0.79	3.85	70.60	18.31	0.51	1.11
Lactopreparata . . . . .	3.28	6.26	22.48	58.89	7.21	0.81	1.07
Liebig's soluble food . . . . .	22.03	0.08	3.32	76.38			1.41
Mellin's food . . . . .	3.93	2.04	11.87	59.45	17.71	0.53	4.47
Milkiné . . . . .	2.74	7.12	13.37	61.19	13.63	0.63	1.32
Nestlé's food . . . . .	2.37	4.94	11.04	43.75	35.73	0.47	1.61
Nursing meal . . . . .	10.84	2.36	6.22	44.66	32.96	0.63	2.31
Nutrico food . . . . .	11.87	4.38	13.40	9.75	57.83	0.61	1.77
Ridge's food . . . . .	8.87	1.67	13.37	8.32	66.35	0.81	0.61
Wagner's infant food . . . . .	5.07	10.91	14.81	37.91	28.91	0.37	2.01
Wells, Richardson & Co.'s lactated food . . . . .	2.94	2.67	13.22	28.84	48.45	1.37	2.51
Zimmerman's health food . . . . .	6.79	1.33	11.16	14.73	63.97	0.71	1.31

(Bulletin U. S. Department of Agriculture, modified by the author.)

infant foods, made up of cereals dextrinized or unchanged, are given in solution, and are very well borne. After the ninth month infants need some cereal added to the milk. In such cases not only is barley or some infant food which is a cereal well borne, but the cereal aids in breaking up the casein, which is at this period given to the full amount present in cows' milk. Infants whose movements are not satisfactory on milk alone do very much better if a cereal is added to the milk at this time.

**Dextrinized Gruels as an Infant Food.**

Chapin in this country and Keller in Breslau have lately advocated the addition of dextrinized cereals to milk in order to facilitate the digestion of the casein. Instead of using water as a diluent, Chapin adds these dextrinized gruels to the milk for both healthy and sick infants. Keller has advocated the use of these gruels with sick infants, especially those of the marantic type.

The majority of pediatricists use no other diluent for the milk than water. We shall therefore only elucidate this method of infant feeding as it applies to sick infants. These mixtures are dextrinized; in this they differ from the former barley-flour mixture of Jacobi, in which a simple dilution of barley was made in water. This was used as a diluent. In the present method diastase, either pure or in



the form of a malt extract containing diastase, is added to the cereal solution. Chapin takes a tablespoonful of flour, adds this to a pint and a half of water, and boils the mixture for fifteen minutes. He then adds a teaspoonful of a solution of diastase. The gruel becomes thin and is dextrinized. It is added to the milk as a diluent in the quantity required.

Keller utilizes the old formula of Liebig in making a malt extract. To this malt extract potassium carbonate is added as a normal salt. 100 grammes of this malt extract are added to 500 grammes (1 pint) of water and dissolved. This is solution No. 1. He then suspends 50 grammes of wheat flour in 500 c.c. of milk. The wheat flour and milk solution is strained. It is then added to the malt extract solution, and both are slowly brought to a boil, being stirred constantly over a slow fire. The mixture is put up in bottles each containing six ounces, corked and kept cool. This mixture contains the dextrinized cereal, malt sugar (the most assimilable form of sugar for the infant), and the proteids of the milk. Liebig malt extract, used by Keller, contains: maltose, 57 per cent.; dextrin, 12.4 per cent. Wheat contains 66.8 per cent. of starch, 7.5 per cent. of dextrin, and a small amount of dextrose. By the action of the ferments in the malt extract the starches are converted into sugars. In this manner a number of easily assimilable and easily absorbable substances are introduced into the economy. The action of these processes on the casein coagulation seems to favor its assimilation.

I am not prepared to pass a final opinion on the merits of this method of feeding. I have tried it in about fifty cases of subacute and chronic enteric catarrh, and have seen infants thrive and increase in weight on this food. In subacute enteric catarrh, in which milk in simple dilution is not assimilated, this food will be well borne and the diarrhoea will subside. Cases of infantile marasmus in older infants and young children improve on this form of food, when the simple milk modifications of Rotch fail. Keller has found that the acid intoxication which exists in the gut of these marantic infants is neutralized by this food. The increased ammonia in the urine of these infants is an index of this form of gut poisoning. This ammonia diminishes or disappears from the urine on the administration of these dextrinized gruels. Keller has given these infants malt extract without cereals, but failed to obtain any increase of weight. I have found that the cases best adapted to the use of this food are atrophic infants from six to seven pounds in weight, too old for a wet-nurse; also infants who after the twelfth month either refuse to take milk food in any form or do not thrive and are stationary in weight. After increasing in weight and taking the foods for two or three months, it is best to wean the children gradually from the food and to accustom them to simpler milk modifications



and other articles of diet. I have experienced no difficulty in accomplishing this. Infants if kept too long on this malted food will develop scurvy.

#### Other Foods.

**Barley-water.**—Barley-water is used as a diluent with normal infants and in forms of diarrhœa. With older infants barley may be utilized in the form of a pap, as may any other cereal.

*Barley-water* is made as follows: A teaspoonful of Robinson's patent barley is added to a pint of cold water in a saucepan. The barley is well dissolved. It is placed on a slow fire and stirred until the whole becomes clear and of the consistency of thin starch paste. Older infants take the barley in much more concentrated form with relish.

**Albumin-water.**—Albumin-water is utilized chiefly in cases of acute stomach and intestinal disorder in which some nutritious and easily assimilable food is needed; albumin-water is then very useful. The white of one egg is dissolved in 8 ounces or a pint of water which has been boiled and then cooled. The solution is strained.

**Peptonized Milk.**—This is milk in which partial or complete digestion of the casein has been accomplished by the addition of peptonizing extracts (*Ex. pancreatis*) and soda in dry form. These peptonizing tubes are sold in the shops (Fairchild's). Milk thus prepared is called peptonized milk or humanized milk. The use of these forms of food can at best be only temporary. As a rule, the children dislike the taste, which is bitter. In other cases the infants do not increase in weight under its use, and the atrophic conditions are perpetuated. I have utilized these peptone preparations but little. Some of my cases have taken the foods thus prepared for a time, and then have had to be fed on other milk modifications.

**Koumyss and Matzoon** have but a very limited field in infant feeding.

**Expressed Beef-juice.**—This food is very useful in forms of diarrhœa and dysentery. A half pound or a pound of chopped lean meat is made into an oval flat mass, placed on the broiler and browned. The juice is then expressed with a small meat press mixed with equal parts of barley-water and salted to suit the taste.

**Acorn Cocoa.**—Acorn cocoa is a preparation made in Germany, and is for sale in the shops. It is useful in cases of diarrhœa and intestinal diseases in which it is advisable to suspend the use of milk, and may be given for days. It contains fat, nitrogenous matter, and tannic acid. A teaspoonful is dissolved in 8 ounces of cold water. The preparation is given in the same manner as milk.

### Feeding of Breast-fed Infants and of Bottle-fed Infants after the Sixth Month.

Camerer has shown, that the secretion of the breast-milk reaches its highest limit in quantity and quality in the sixth month of lactation. In many cases it then diminishes in quantity and quality. If the infant gains steadily after the sixth month, nothing additional is given. If, however, the increase of weight is not satisfactory, we may at this period begin the daily administration of one or two bottles of modified cows' milk up to the ninth month, the time when the baby is weaned. On the eruption of the incisor teeth the baby, generally at the seventh month, is allowed a cereal in the shape of some prepared barley or cracker or rusk (*Zwieback*) to nibble upon once a day. The barley is omitted if the infants are inclined to be constipated. I find that one rusk (*Zwieback*) or cracker daily is sufficient. As to cereals, the same procedure is followed with the bottle-fed infant after the teeth have appeared or after the seventh or eighth month.

### Feeding from the Ninth to the Twelfth Month.

**Breast-fed Infants.—Weaning.**—It is not advisable to wean infants at the outset of the summer season, even though they must be kept at the breast a few months longer. The infant must not be wholly deprived of the breast in the warm season. If the bottle milk disagrees with the baby, it will go very hard with it should weaning have been accomplished at the outset of the summer. It takes about eight weeks to wean a baby completely. If the baby has had the benefit of one or two additional bottles daily from the sixth month, the process is comparatively simple. If, however, the infant has been kept on the breast exclusively until the ninth month, weaning is often very difficult. The infant will not take the bottle so long as there is a breast at its disposal. The only way out of the difficulty is to dispose of the nurse and thus force the infant to take the bottle. This requires much moral courage on the part of the physician. In those cases in which the mother nurses the baby we cannot always gain her co-operation in denying the breast to the infant. The difficulties of weaning in such cases will be great. In weaning, I give those modifications of cows' milk which contain from 1 to 1.2 per cent. of proteids and from 2 to 2.5 per cent. of fats until the infant is fully weaned. I then increase the strength of the milk to that given to the bottle-fed baby at the ninth month. The bottle-fed baby at this time is given almost pure milk. It is well to mix the milk with a small quantity of water (1 ounce of water to 7 ounces of milk). In addition, from the ninth to the twelfth month both breast-fed and bottle-fed infants are given cereals in the shape of pap, barley, or granum, or rusk (*Zwieback*) or crackers



twice daily. Some mothers give these infants an ounce of expressed beef-juice with barley once a day. Infants relish this change.

#### Feeding from the Twelfth to the Eighteenth Month.

From the twelfth to the eighteenth month I allow the infant the following dietary :

*Milk*, a quart and a half pint to a pint daily.

*Cereals*. Rusk (*Zwieback*) or crackers, two of each a day. Sponge-cake in shape of long sugared slices. *Barley, granum, oatmeal*, strained, in form of pap, once a day.

*Eggs*. One soft-boiled egg a day.

*Beef-juice*, expressed, with barley-water.

The above is divided up into five meals daily.

#### Feeding from the Eighteenth Month to the End of the Second Year.

Five meals in the twenty-four hours, consisting of :

Milk, one quart (outside limit).

Eggs, soft-boiled, one or two daily.

Soup or beef-juice.

Meat, beef, about 2 ounces.

Vegetables.

Cereals.

*Milk*. Some children will take more or less, some very little milk at this period. The eggs are boiled for two minutes. Most children will require only one a day.

*Soups*. The quantity of beef-juice or soup should not exceed 4 ounces.

*Meats*. The ordinary boiled meat, inside of a lamb chop, small piece of steak, roast beef, chicken. Gamey meats and fat meat, such as mutton, ham, pork, are to be avoided.

*Vegetables*. Potatoes, peas, beans, carrots ; all should be given in purée form.

*Cereals*. Barley, rice, granum, wheatena, oatmeal, rusk (*Zwieback*), crackers of all kinds, cocoa, farina.

*Fruits*. Orange (juice), ripe apples, and pears.

*To be avoided*. Vinegar, cabbage, salad, coffee, tea, wine, spices too great an amount of amylacea.

A dietary from the eighteenth month to the end of the fourth year might be formulated as follows :

First breakfast, 8 A. M. : 250 c.c. (8 ounces) of milk ; 60 grammes (12 ounces) of bread or crackers.

Second breakfast, 10.30 A. M. : 180 c.c. (6 ounces) of milk 1 rusk (*Zwieback*) ; juice of orange.

Dinner, 1 P. M. : 120 grammes (4 ounces) of soup ; 75 grammes (2.5 ounces) of meat ; vegetables.



Afternoon lunch, 4 P. M. : 250 c.c. (8 ounces) of milk or cocoa ; rusk (*Zwieback*) or cracker.

Supper, 6.30 or 7 P. M. : soft egg ; 250 c.c. (8 ounces) of milk ; cracker, toasted bread, or farina in milk.

*Candy.* I allow one or two pieces of candy, generally good chocolate, daily to older children.

From the third to the sixth year of life the diet should be mostly fluid or semifluid. The basis of all such diets should be milk, milk soups, eggs, meat, butter, cocoa, breadstuffs, vegetables, and fruits. The number of meals a day should be five.

The following is a schedule of a liberal diet at this time :

First breakfast, 8 A. M. : 330 c.c. (11 ounces) of milk ; 100 grammes (3.5 ounces) of bread.

Second breakfast, 10.30 A. M. : 330 c.c. (11 ounces) of milk ; 90 grammes (3 ounces) of bread ; 10 grammes ( $\frac{1}{3}$  ounce) of butter ; juice of orange.

Dinner, 1 P. M. : 180 c.c. (6 ounces) of soup ; vegetables ; 90 grammes (3 ounces) of meat.

Afternoon lunch, 4 P. M. : 330 c.c. (11 ounces) of milk ; 90 grammes (3 ounces) of bread.

Supper, 7 P. M. : 250 c.c. (8 ounces) of milk or mixed with 60 grammes (2 ounces) of cereals.

This is a liberal diet. Some children will not take as much milk as is here prescribed. Eggs have not been included, nor certain additional fruits which it may be allowable to give. This form of diet, with some slight modifications, is suitable up to the tenth year of life. The main object of all dietaries, after the eighteenth month, is to mix the carbohydrates, fats, and albuminoids in rational proportions. The following table, by Camerer, shows this distinctly :

Age and weight . . .	Second to fourth year. 12.7 kilo. <sup>1</sup>	Five to six years. 18.7 kilo.	Seven to ten years. 24 kilo.
Total food (daily) . .	1183. grammes.	1517. grammes.	1699. grammes.
Albumin . . . . .	46.	64.	67.
Fat . . . . .	39.	46.	32.
Carbohydrates . . . .	117.	197.	251.
Water . . . . .	957.	1200.	1333.

### The Feeding of Sick Infants and Children.

The feeding of sick infants is considered under the headings of the various diseases. It must always be borne in mind that infants and children, if left to their own resources, would take either very little nourishment or too much. In certain marantic conditions infants will take very large quantities of food if it is given to them. The infant's cries are interpreted by the mother as being due to hunger, when they may be due to colic or intestinal

<sup>1</sup> Kilo equals 2.2 pounds.

distention. In these cases the mother gives too great a quantity of food, and the infants suffer from dilatation of the stomach. In typhoid fever, pneumonia, or other acute disease the patient, if fed at long intervals, takes but little food. I am in the habit of giving such infants or children small quantities at short intervals. If the infant takes a small quantity at each feeding, the aggregate amount in twenty-four hours is sufficient to maintain nutrition.

After operations, such as those for empyema, infants and children must be carefully and systematically fed up in order that they may combat the ravages of disease. The necessity of careful feeding is seen in typhoid fever in the fifth and sixth weeks, at which time there is great emaciation and the temperature has dropped to the normal. If we fail to feed up the patients, they remain emaciated and show slight inanition temperatures. On the other hand, we must not give large quantities of indigestible food. We must choose the foods carefully. Convalescents can take in twenty-four hours much larger quantities of food than the normal, healthy child. The quantity given at each feeding should be smaller than in health. The nitrogenous foods, such as milk and eggs, and also sugars, starches, and cereals of all kinds, are easily assimilable. Alcoholics, when given, should be well diluted. Rectal feeding is contraindicated in diarrhoeal conditions and states of rectal intolerance. On the other hand, if the stomach rejects food repeatedly it is well to give that organ complete rest. Under such conditions even water is not introduced into the stomach. The patient is fed for twenty-four hours or more per rectum.

#### LEADING AUTHORITIES QUOTED IN CHAPTER I.

- Ahlfeld*: Ueber d. Ernährung. d. Säugl., etc., 1878.  
*Alix and Vierordt*: "Temperatures," Gerhardt's Hand-book, vol. i.  
*Backhaus*: "Milch Analyse," Berlin. klin. Wochenschr., 1895.  
*Boginsky, A.*: Antipyrese, etc., Berlin, 1901.  
*Budin, P.*: Le Nourisson, Paris, 1900.  
*Comerer, W.*: Der Stoffwechsel des Kindes, etc., 1894.  
*Cantani*: "Cholera Behandlung," Berlin. klin. Wochenschr., 1892.  
*Chapin, H. D.*: N. Y. Med. Jour., February, 1901.  
*Conrad, F.*: Die untersuch der Frauenmilch, 1880.  
*Doane and Price*: Maryland Agricul. Bull., 77.  
*Eröss, Julius*: Ueber die Sterblichkeit Cong. Hygiene, 1894.  
*Escherich*: "Morbidity," etc., Jahrbuch. für Kinder, dritte folge, Bd. i. Heft 1  
*Epstein, A.*: "Cholera Infantum," Hensch Festschrift, 1890.  
*Fehling*: "Form des Beckens," Arch. f. Gyn., Bd. x.  
*Fourmier*: Syphilis Hered. tarda. 1886.  
*Herz, P.*: Dissertation, Klin. untersuch. neugeb., 1900.  
*Keller*: Malzsuppe, Jena, 1894.  
*Knöpfelmacher*: Ernährung mit Kuhmilch, 1894.  
*MacDonald*: "Experimental Study of Children," U. S. Bureau of Education, 1899  
*Meigs, A. V.*: Milk Analysis and Infant Feeding, Philadelphia, 1885.  
*Preyer*: Die Seele des Kindes, Leipzig, 1890.  
*Schlichter*: Untersuch. u. Wahl der amme, Vienna, 1894.  
*White and Ladd*: Phila. Med. Jour., February 2, 1901.  
*Wroblewski, A.*: Beitrag zur Kennt. des Frauen Caseins, 1894.



## CHAPTER II.

### PREMATURE INFANTS—DISEASES OF THE NEWBORN INFANT—INJURIES INFLICTED DURING BIRTH.

#### PREMATURE INFANTS.

PREMATURELY born infants may be congenitally weak, although they are not always so. The organs, and especially the lungs, are unformed and their functions are incompletely performed. The body is spare or emaciated; the skin is soft, delicate, uniformly red, and transparent, showing the bloodvessels. The infant does not cry, but rather whimpers; the respiration is scarcely perceptible. The thorax does not move, and there is marked muscular inertia. The limbs hardly move; the infant lies in a torpid condition. It often does not swallow its food. The heart-beat is feeble; there may be œdema of the extremities; and the intestine and stomach are easily irritated. The liver performs its functions imperfectly, giving rise to icterus. The delicacy of the skin exposes it to irritation, with resulting formation of erosions and sclerema. The temperature in the rectum is below the normal, and ranges from 86° to 95° F. (30° to 35° C.).

**Management of Premature Infants.**—The treatment of a premature infant born asphyxiated is at first much the same as that detailed in the section on Asphyxia of the Newborn. In most cases our efforts should be directed toward maintaining the body temperature, nursing the infant properly, and stimulating the heart and respiration. With some infants at the eighth month little more is necessary than to wrap them up snugly and maintain the surrounding temperature at the necessary elevation by means of warm bottles. In most cases the task of constantly maintaining a temperature of from 86° to 98.6° F. (30° to 37° C.) is not easy unless resort is had to an incubator. That this maintenance of an equable high temperature is of importance has been shown by Schmidt. This author proved that loss of body weight could be brought about by rapid changes in the temperature of the atmosphere surrounding the infant.

**Incubators.**—The most efficient incubators are made of metal or are porcelain-lined, are simple in construction, and allow of thorough ventilation while maintaining the desired temperature. Infections being common, the incubator should be so constructed that it can be easily cleaned and subjected to sterilization before use. Wooden



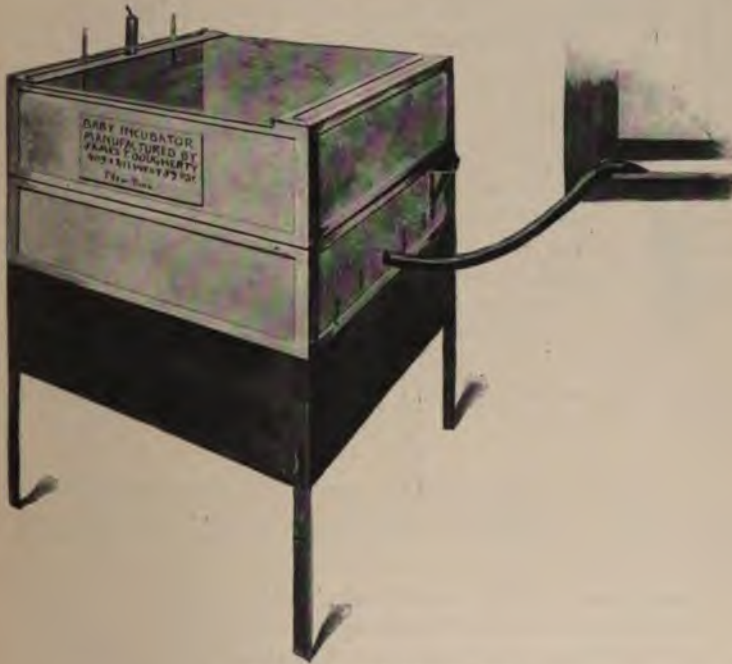


The *indications* for the employment of any form of incubator are : (a) Prematurity, the infant weighing 2000 grammes or less. On the other hand, infants weighing 1800 grammes can sometimes if strong be reared without an incubator. (b) Subnormal rectal temperature. (c) Cyanosis or sclerema.

The temperature of the interior of the incubator is regulated by that of the infant. If that of the infant is  $86^{\circ}$  to  $89.6^{\circ}$  F. ( $30^{\circ}$  to  $32^{\circ}$  C.), that of the incubator should be  $95^{\circ}$  to  $98.6^{\circ}$  F. ( $35^{\circ}$  to  $37^{\circ}$  C.).

The infant in any incubator should increase regularly in weight and strength ; it should have two movements daily, and take its

FIG. 24.



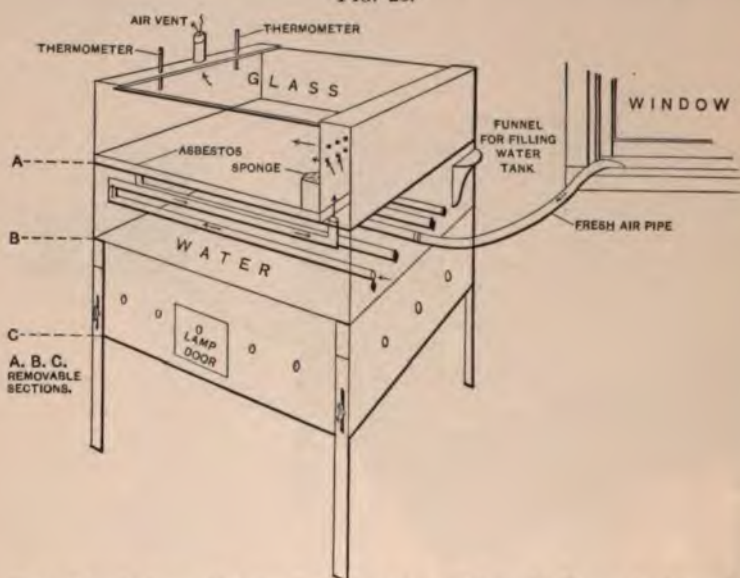
Simple form of baby incubator.

nourishment at regular intervals. If it loses in weight, remains cold, cannot be roused, breathes superficially, develops cyanosis, dyspnœa, diarrhœa, cough, or vomiting, the outlook is grave. Even if the infant is thriving, it should not be allowed to remain torpid. If the respiratory movements are shallow, the infant should be taken cautiously out of the incubator and from time to time caused to cry. In this way the lungs will expand and become aërated. The infant should be turned on its side and kept lying

in that position. In this way hypostasis in the lower and posterior part of the lungs is avoided.

The feeding of the premature infant is a difficult problem. If the infant is very young it may not be able to grasp the breast. It must then be fed with a pipette or a nursing tube constructed for the purpose (Fig. 26). In such cases the milk is pumped from the breasts and transferred to the infant. If fed with cows' milk, the dilution should contain a low percentage of proteids (0.5 per cent.) and fat (to 1.5 per cent.). The quantity to be given at each feeding should

FIG. 25.



Plan of simple form of incubator. A, B, water tank heated by lamp; in this tank are the fresh-air pipes. The air is heated before passing into the top section in which the infant lives.

not at first exceed a half ounce, and should gradually be increased until the infant arrives at the term age.

The **prognosis** varies with the period of prematurity of the infant. At the sixth month about 30 per cent., at the seventh month 63.7 per cent., at the eighth month 85 per cent., and at eight and a half months 90 per cent. are saved (Bertin). These figures, of course, are only approximate, and will vary largely with the care taken with the incubator and the absence of accidental infections. For this reason it has been proposed by Bosi, Giudi, Escherich, and others, to construct incubator wards, in which the infant shall not be exposed to changes of temperature and to danger of infection when taken out of its cradle.

The most frequent cause of death is infectious bronchopneu-



monia. In such cases percussion rarely establishes dulness of any extent, the respiratory sounds are feeble, air scarcely enters the lung, cyanosis is present, and the temperature may be even subnormal. There may be eruptions on the skin, and death may take place with partial or general convulsions.

**Morbid Anatomy.**—Post mortem are found areas of bronchopneumonia with atelectasis. On the surface of the lungs are hemorrhagic areas resembling infarctions. There is hemorrhagic pneumonia. The bronchial nodes may be enlarged, and there may be pericarditis. The intestines, liver, and kidney present lesions similar to those found in sepsis. The hemorrhagic pneumonia is due to infection by streptococci, staphylococci, *Bacillus coli communis*, and pneumococci (Mussy, Labi, and Levi). These infections may be local and limited to the lung, or general. The sources of infection must be sought in lesions of the skin, mucous membrane, and respiratory passages transmitting noxious elements from the air, dust, or objects brought in contact with the infant's hands, linen, and food. The prophylaxis is therefore clearly indicated. The skin must be kept clean and the mouth protected from traumatism and infections. The food, preferably the breast, must be unirritating. The hands of the nurse must be scrupulously clean, and the contents of the diapers removed carefully and quickly.

FIG. 26.



Breck's feeding tube for premature infants.

## ASPHYXIA OF THE NEWBORN INFANT.

Asphyxia is a condition produced by interference with the oxygenation of the blood. In the uterus respiration is effected through the placenta. As soon as the placenta is separated in part or as a whole from its uterine attachments the disturbance in the circulation causes efforts at respiration as a result of dyspnoea. If the placenta is separated prematurely, there are consequent efforts at respiration, during which liquor amnii and mucus may be aspirated and asphyxia thus produced.

In the extra-uterine form of asphyxia the infant is born and makes efforts at respiration, but inherent constitutional weakness, weakness of the respiratory muscles, or deformity or disease of the lung render full expansion of the lung impossible.

**Morbid Anatomy.**—The blood in infants who have died asphyxiated is thin and fluid. The right heart and large vessels are filled with blood, as are also the sinuses of the dura mater, pia mater, and

liver. The liver is dark and bluish in tint. Punctate hemorrhages are found in the pia, pleura, pericardium, peritoneum, liver, kidney, retroperitoneal connective tissue, uterus, kidneys, suprarenal capsule and retina. There is a serosanguinolent effusion in the cavity of the peritoneum, pleura, and pericardium. Edema of the extremities, scrotum, and connective tissue about the umbilical vessels and perineum, is present. The lungs are dark red and heavy. Ecchymoses are seen underneath the pleura and pericardium. In the lungs are islands of aerated tissue; also areas of atelectasis, even though the infant has breathed. Trachea and bronchi may be filled with liquefied amnii, mucus, or meconium. These may be found also in the smallest bronchi. The stomach may be filled with air or meconium.

**Symptoms.**—If in a normal state when born, the infant breathes energetically, cries lustily, and opens its eyes, and the skin, which is of a purple hue at first, rapidly assumes a pinkish tint. If asphyxia be present, however, we may have two sets of symptoms, which are characteristic of two forms of this condition.

In the first form, or early stage, of asphyxia, the skin has a bluish or pinkish-blue tint. The face is swollen and the conjunctivae injected. The infant does not move the extremities. The musculature retains its tonicity; the heart is slow but forcible; the apex beat is apparent to the eye; the vessels of the cord are filled with blood and pulsate; the respiratory efforts may be shallow and infrequent, or absent; the infant can be roused and caused to cry.

In the more advanced form of asphyxia the face is pale and waxy, the lips cyanosed; the extremities hang lax, and the muscular tonus is absent; the head falls to one side and the jaw drops. There is no attempt at respiration or only imperfect gasping efforts. The infant has a corpse-like appearance. The heart-beat is weak though palpable. The vessels of the cord are collapsed and pulsation is weak. If a few gasps of respiration are made at birth, these soon cease. On attempt at respiration the ribs are retracted, but the muscles of the face are immobile. Air is prevented from entering the lung by the inspired mucus. Reflex reaction is absent; there is no response to irritation. If untreated, infants in this stage of asphyxia die. If they live, efforts at respiration must be encouraged, else the infants relapse into a stupid condition and the respirations become superficial.

**Diagnosis.**—Asphyxia must be differentiated from the effects of pressure due to cerebral hemorrhage occurring at birth in the course of prolonged labor or application of the forceps. In a large hemorrhage death is rapid, but in slight hemorrhage it may be difficult to make a differential diagnosis. If there is a hemorrhage on the surface of the brain, the symptoms may closely resemble those of asphyxia. The breathing is very superficial; the infant lapses into sopor; the pulse may at first be slow and subsequently rapid. There



may be occasional convulsions. Only the subsequent history will clear up these cases. Asphyxia may be combined with cerebral hemorrhage. The history of the birth as to the use of forceps and the duration of the labor will aid us. If after irritation the infant relapses into sopor, if the pulse continues slow and there are repeated convulsions, we may assume hemorrhage.

The **prognosis** in all forms of asphyxia if untreated is grave, and in the second stage is necessarily fatal. If treated, however, the majority of these cases recover, especially those in the first stage. As to the cases of the second stage, much will depend on the duration of the second stage of labor and the compression of the cord. The cases in which cerebral hemorrhage of any severity is combined with the asphyxia are grave. Little and Mitchell have demonstrated that idiocy may subsequently develop in these cases.

The **treatment** of asphyxia is directed to clearing the air-passages as much as possible of obstructing mucus, increasing the number of respirations, and stimulating the circulation. The mucus and aspirated meconium are immediately removed from the mouth by introducing the finger quickly but gently. If râles are heard in the chest, a soft-rubber catheter is introduced as far down as the superior opening of the glottis, and as much mucus as possible is removed by mouth suction. Introduction of the catheter into the trachea is hardly necessary. Special instruments are not always at hand, and the catheter is equal to all emergencies.

In order to stimulate the surface, the infant is quickly placed in a bath at 105° F. (40.5° C.), and then in a cold bath, thence transferred to a warm blanket and rubbed thoroughly dry. After this the infant is, if possible, roused by striking the buttocks quite sharply. If these methods do not cause the infant to cry and breathe deeply, artificial respiration by the Schultze method should be resorted to. This method is so well described in all obstetric treatises that it needs only brief mention. The operator, standing with his body well balanced, grasps the infant by the shoulders, the thumbs being on the anterior aspect of the thorax, the index fingers in the axillæ, and the other fingers on the back of the chest. The head is supported by the ulnar side of the wrists. The operator allows the infant to hang from his hands down between his legs. The infant is then raised or swung upward above the level of the operator's head to the vertical, so that the lower part of the trunk of the infant is bent on the thorax. The thorax is thus compressed, causing passive expiration. The infant is held for an instant in this position, and then swung down to the original hanging position. Passive inspiration is thus performed. This manœuvre is repeated about sixteen to eighteen times a minute. During the manœuvres the bronchi and mouth are freed from mucus, meconium, and liquor amni, if present. The Laborde method is that by which



traction on the tongue is made ten or twelve times a minute. The Dew method seeks to accomplish the same result as the Schultze method by simpler means. The infant is grasped by one hand at the nape of the neck, and by the other hand at the knees. The thighs rest in the palm of the hand. The thorax is flexed on the abdomen, and then extension is performed. Alternate expiration and inspiration take place. Inflation of the lungs by means of instruments introduced into the larynx is dangerous. There are other methods of artificial respiration which may be resorted to, such as the Marshall-Hall method, but, on the whole, the method of Schultze seems the most effective. The danger in all cases is in abandoning efforts at resuscitation too early. So long as the heart is beating we should continue our efforts. After the infant has been brought out of the stage of severe asphyxia there is always danger of relapse into a soporous state. In this condition flagellation on the buttocks at regular intervals may be necessary for days at a time. After being worked over for days, such infants may die and show extensive atelectasis in spite of the fact that respiration has occurred.

### ASPHYXIA SUBSEQUENT TO BIRTH.

In these cases there is no disturbance of the placental circulation previous to the birth of the infant, and therefore no asphyxia. Asphyxia appears after birth as a result of some abnormality in the respiratory apparatus, or of disease of the lung, such as syphilitic hepatization; of pleural exudate; of compression of the air-passages by a struma; or of defects of the diaphragm, or deficient development of the lungs. In some cases there may have been injury or compression in the vicinity of the respiratory centre.

Prematurity of the infant carries with it a soft condition of the ribs and weakness of the respiratory muscles, an insufficient development of the respiratory centre, and foetal atelectasis, which give rise to a state of asphyxia. The more premature the infant the more pronounced are these conditions.

**Symptoms.**—A premature infant makes no decided effort at respiration after birth. Inspiration is absent or is hardly noticeable and shallow. Râles are absent. The vessels in the umbilical cord are filled with blood and pulsate distinctly. The heart has a normal frequency at first; then the contractions become slower and may eventually be increased in frequency. The skin is bluish-red in color; the extremities cool. If there is any disease or deformity of the lung, the infant dies soon after birth. These cases are only of scientific interest. Of more importance to the physician is the premature infant normal in all respects save in the fact of its expulsion from the uterus before term.

Premature infants at the sixth, seventh, or eighth month are not all born debilitated, nor are all debilitated infants necessarily premature. There are infants born at the eighth month which are as easily reared as an infant at full term.

### ATELECTASIS OF THE LUNG.

This condition has been referred to in the sections on Asphyxia. Atelectasis, or collapse of the lung, may be congenital or acquired. In the congenital variety the infant is either weakly or born prematurely. The respiratory muscles do not possess sufficient tonus to inflate the lung. The result is that the lung remains in the collapsed foetal state. In the acquired form, the result of obstruction of the bronchi or alveoli, compression of the lung by an exudate in the pleura, deformity of the vertebral column, or aneurism of the aorta, the lung cannot expand. The lung at birth is compact, the alveoli being collapsed. The respiratory efforts inflate the alveoli. In atelectasis the alveoli can be inflated post mortem. Any inflammation of the smaller bronchi subsequent to birth may prevent inflation of the alveoli, and thus cause atelectasis. Any atelectatic area may become inflamed (bronchopneumonia), and subsequently involve other alveoli. Therefore, in the same lung areas of atelectasis and bronchopneumonia may be present, though distinct from each other and not necessarily in causal relationship. In such lungs atelectatic areas are seen interspersed with inflated areas. The collapsed areas are depressed beneath the surface, are of a dark-red or bluish color, and are beefy on section.

The **symptoms** of atelectasis are not always clearly defined. As a rule, the infants if premature are weak; their torpid state has been described in the section on Asphyxia. On the other hand, should atelectasis develop some time after birth as a result of inflammation and plugging of the smaller bronchi, we shall have the combined physical signs of atelectasis, bronchitis, and possibly bronchopneumonia. In this last class of cases the physical signs are as follows:

**Inspection.**—There is intense dyspnoea; the lower ribs are retracted, and the efforts at inspiration are labored and move the upper part of the thorax less than the lower portion. The surface is pale and sometimes cyanosed. Efforts at coughing are ineffectual, and may bring up a frothy, clear expectoration which adheres to the lips. Sometimes the breathing is quite irregular and catchy, or very shallow; at times the infant seems to cease breathing.

**Palpation** with the palms of the hands is negative except where râles are abundant, when a fine fremitus is present. There is little or no vocal fremitus; it may be increased, or it may be diminished, especially in areas designated vesiculo-tympanic.



**Percussion** reveals distinct small areas of dulness with a tympanic note, slight dulness, or marked dulness, especially if areas of collapse are present with pneumonia. Sometimes the note over the rest of the thorax, behind especially, is vesiculo-tympanic. At times when the areas of collapse are small no dulness is elicited.

**Auscultation.**—In areas generally situated at the apex or toward the base of the lung the air does not seem to enter freely on inspiration, and the expiratory sound is hardly audible (collapse of area) or absent. Breathing is otherwise puerile or exaggerated, rarely bronchial.

In various parts of the lung are heard very fine subcrepitant râles. Crepitant râles are very distinctly heard in other areas, and are distinguished from the coarser subcrepitant râles by their fine quality. Areas of pneumonia can thus be recognized by the fine crepitations, by the atelectasis, and by the absence of respiratory sounds and dulness. Voice sounds vary greatly. When the infant cries the vocal resonance may seem increased, and again normal; or if the pneumonic area is extensive and is in the vicinity of a large bronchus, we may have tubular resonance.

Temperature is often normal or subnormal; later, it may be elevated.

Convulsions are common in atelectasis—in fact, they are peculiar to the disease. They are repeated at frequent intervals, and an infant may have three or four attacks of general convulsions in the course of the twenty-four hours. At the onset of the convulsions the cyanosis increases.

The **diagnosis** of post-natal (acquired) lobular atelectasis will depend upon:

*Convulsions.*—Given the case of a newborn infant delivered without forceps or force, in the absence of signs of any other disease the presence of repeated convulsions, with cyanosis and dyspnoea in the intervals, should make us consider the possibility of atelectasis.

*The presence of areas of slight dulness*, or tympanic dulness, or vesiculo-tympanic resonance all over the chest.

*Fine subcrepitant râles.*

*Still finer crepitant râles.*

*Areas in which the air enters incompletely.*

**Treatment.**—The treatment must be directed toward stimulating the heart and increasing the respiratory efforts if the infant is weak or premature. If the heart is weak, the treatment is much the same as in bronchopneumonia. If the infant does not breathe satisfactorily, it is well to make it cry vigorously several times in the twenty-four hours, so that the collapsed area of lung may be inflated and the mucus in the bronchi expelled. Unless made to cry, these infants lie torpid and hardly seem to breathe. The areas of atelectasis are thus increased. If the temperature is subnormal and the infant seems



chilled, we may stimulate it by the application of heat externally, either by means of warm baths, hot-water bottles, or an incubator.

### SEPTIC INFECTION OF THE NEWBORN INFANT.

By septic infections are meant certain general phenomena produced by bacterial toxins or toxalbumins, or by the entry of bacteria themselves into the body by way of the blood-channels or lymphatics. The newborn infant is particularly susceptible to infection. At this period of life the natural means of defence are lacking. The lymph-nodes and spleen are undeveloped, and as a result phagocytosis, the chief protection of the adult, is absent. The skin also is in a very vulnerable state. It is a ready means of entrance for bacteria, as are also the mucous membranes (Epstein). The lack of febrile reaction also demonstrates the fact that in the newborn there is little resistance against invasion of bacteria. With our present incomplete knowledge, we class as septic infections such conditions as diarrhoeas, bronchitis, pneumonia, hemorrhagic conditions, Winckel's and Buhl's disease, and dermatitis exfoliativa.

**Etiology.**—The most frequent causes of septic infection are the pyogenic bacteria, the streptococci and staphylococci. Following these in order of importance are the bacilli of the coli group, the pneumococci, bacilli of general hemorrhagic infection (Babes), the *Bacillus pyocyaneus* (Neumann), the capsule bacillus of Dungen, the *Bacillus enteritidis* (Gärtner), found in hemorrhagic affections resembling Winckel's disease, and the bacillus of Finkelstein, found also in a hemorrhagic condition. The bacteria exist in the air of hospital wards (Emmerich, Babes, Gärtner, Prudden). They are found in the normal breast milk (Neumann) and in the milk of breasts which are the seat of ulceration, fissure, or abscess. The body of the mother, the lochia, and also the liquor amnii after rupture of the membranes, are all sources whence bacteria may gain access to the newborn infant. As a rare source of infection may be mentioned the incubator in which septic cases have been nursed (Allard). The bath water has been the means of spreading epidemically in institutions dermatitis exfoliativa (Fischl) and Winckel's disease (Winckel).

The newborn infant possesses in its own body sources of infection. Thus it may infect itself (auto-infection) through the secretions of its respiratory passages and through the stump of the umbilical cord, in which even in the healthy infant pathogenic bacteria may be found.

Bacteria or their toxins may gain access to the body through the intact or wounded skin, the umbilicus, the mucous membranes (buccal or pharyngeal), through the lungs in the respired air, through the digestive tract by means of the food, through the conjunctivæ and the ears, and finally through the genital tract.

**Symptoms.**—It is almost impossible to particularize any forms of sepsis so far as the general symptoms are concerned. The reaction in the newborn infant is so imperfect and the signs are so equivocal, that it is often only at the autopsy table that the nature of the lesion is determined. It will be convenient, therefore, simply to enumerate the objective changes noted in the various structures of the body in this disease.

The **skin** may be dry, or the seat of localized œdema or sclerema. It may be the seat of erythema of a polymorphous variety, either on the body or on the extensor surface of the arms or hands. There is sometimes a general or localized cyanosis. A peculiar form of this cyanosis has been described by Finkelstein—the so-called angio-spastic cyanosis—in which a central pallor and peripheral lividity are present in the patches. The cyanosis may be limited to the hands and feet.

Eruptions of a pemphigoid character are sometimes seen in sepsis of the newborn infant. The vesicles may be the seat of supuration, or there may be ulcers and intertrigo varying from superficial erosions to extensive areas of gangrene. The skin may be pale or icteric in hue. There are erysipelatous patches, furuncles, and abscesses of a multiple variety.

The mucous membrane of the mouth is dry and fissured, and the tongue dry and coated. The roof of the mouth is the seat of ulcerations, superficial or deep, occurring at the median raphé, where we find normally Epstein's pearls, or laterally over the hamular processes of the palate bone (Bednar's aphthæ). The mouth may be the seat of pseudomembranous deposit not due to the diphtheria bacillus (Epstein). In these cases of sepsis sprue may engraft itself on the mucous membrane of the mouth and extend to the pharynx, œsophagus, and stomach. The vagina in female infants may be the seat of catarrhal or pseudomembranous inflammation.

**Umbilicus.**—Normally, pathogenic bacteria are found about the stump of the desiccating cord. In septic conditions the cord does not fall off promptly. The tissues about the umbilicus are inflamed and the seat of phlegmon and suppuration. I have seen pus burrow downward toward the bladder along the course of the fetal structures. The bloodvessels of the cord may be the seat of inflammation, as will be shown later. In some forms of sepsis in which the infectious material may have gained entrance through the umbilicus, the latter may show absolutely no change from the normal.

**Joints.**—There may be swelling in the muscles about the joints, as in forms of intramuscular abscess, or the joint itself may be the seat of septic suppuration or so-called osteomyelitis (Plate V.). The shaft of the bone or the epiphysis only may be involved. One or many joints may be the seat of suppuration.

**Nervous System.**—Functional symptoms, such as apathy, rest-





PLATE V.



Sepsis in the Newborn Infant. Suppuration of the right knee-joint. Osteomyelitis of the epiphyses of the bones forming the joint.

lessness, or convulsions, may be present, or there may be localized facial paralysis or paralysis of the extremities, traceable to meningitis or encephalitis. Hemorrhages in forms of sepsis may give rise to paresis simulating the traumatic palsies of the newborn.

**Respiratory Tract.**—The respiratory tract may present catarrhal or pseudomembranous inflammation of the nose, tonsils, larynx, or trachea. The bronchitis and pneumonia, especially in the septic forms of diarrhoea, may be of obscure nature and run an insidious course.

The **heart** may be the seat of septic endopericarditis. This form of pericarditis is rarely diagnosed.

The **digestive tract** is the seat of septic diarrhoea. In the cases described by Fischl and Czerny there was complicating bronchopneumonia of a septic type.

The **liver** may be the seat of enlargement in cases of extended duration, but the spleen is rarely so.

The **urine** in most cases indicates the presence of a diffuse nephritis.

The **body weight** diminishes markedly and rapidly.

The **temperature** is not characteristic. In the severest forms of sepsis it may be normal or subnormal; in other cases there may be a rise of a degree or more. I have seen this in milder cases. A new complication may be ushered in with a rise of temperature, as often happens with older infants and children, but is not necessarily so.

**Morbid Anatomy.**—Lack of space forbids entering into the details of the pathological alterations found in the septic infections of the newborn infant. The changes in the skin have already been described. Those of the umbilicus will be found under the section on Umbilical Infection. The appearances in the mouth, nose, and throat have been described, as well as those of the lungs. The alterations in the gastro-enteric tract are detailed in the chapter on Diseases of the Gastro-enteric Tract.

The liver and kidneys are the seat of parenchymatous or diffuse suppurative changes. The peritoneum is ordinarily intact, although formerly authors believed it to be frequently involved. The pericardium, endocardium, and myocardium may be the seat of slight or marked changes. Microscopical examination of the blood may reveal the infecting bacteria.

**Diagnosis.**—The origin of some cases of sepsis of the newborn infant is so obscure that not only is a diagnosis made with difficulty, but it is not always possible to determine the point of entrance of the infectious agent. In cryptogenetic cases no lesion may be visible. If an infant cries when diapered or when it is washed in the bath, the joints should be examined for suppuration. A pseudomembranous deposit or an ulceration in the mouth is a sign of traumatism



with infection. A diarrhœa in the newborn infant is of serious moment. The umbilicus, if swollen or red, should receive due consideration. In cases in which there is no external lesion a blood-culture should be made to determine the presence or absence of micro-organisms in the blood. Puncture of the spleen for the detection of micro-organisms has been advised. Such a procedure may or may not be advisable, according to the indications present in the case.

**Course and Prognosis.**—Some forms of acute sepsis prove fatal in a few hours. Others, and they are the most common, last from a few days to a week. Finally, the subacute cases, which are complicated with progressive emaciation, diarrhœa, and pneumonia, extend over two or more weeks. Septic osteomyelitis and chronic omphalitis are especially protracted. The prognosis in these cases is always grave. Mild forms of intestinal sepsis, after pursuing a short course with fluctuating temperature, may recover completely.

**Treatment.**—There is no specific for sepsis in the newborn infant. Prophylaxis is of the utmost importance. The hands of the accoucheur must be as clean in handling the newborn infant as in the treatment of the mother. The cord is tied with precautions described elsewhere. The mouth is not washed. As Epstein has pointed out, Bednar's aphthæ and pseudomembranous inflammations are thus avoided. The nasal passages are not inspected more than is absolutely necessary. The bath water should be clean and not below 100° F. (38° C.). The food should receive attention. The infant should not nurse a fissured or an inflamed breast. The breast nipple should be cleaned before and after nursing, as stated in the section on Hygiene. The room in which the child sleeps should be ventilated. Contact with the secretions of the mother (lochia) should be avoided.

Therapeutic measures are directed to combating the symptoms. The strength should be supported, and for this purpose alcohol may be used with small doses of strychnine. The antistreptococcic sera are of doubtful efficacy. The administration of alkalies, such as the salicylate, benzoate, and carbonate of sodium, has been strongly advocated. High saline enemata are advised by Sahli.

### DISEASES OF THE UMBILICUS.

The umbilical cord dries up and drops off in five days, leaving a granulating stump. In the case of weakly infants the cord may not fall off until much later. The stump may become inflamed, and pus may form. This in the majority of cases is due to infection. Infection usually takes place at the time of ligation or before the cord separates from the stump. Gangrene is indicative of infection. The appearance of the stump in omphalitis varies. In some cases



the inflammation is slight, but in others the tissues are red, infiltrated, and coated with necrotic masses resembling pseudomembrane. Numerous small abscesses may be present. The great danger is that the process may involve the umbilical vessels. If the inflammation remains local, recovery is the rule. If the vessels become involved, sepsis may result.

**Therapy.**—Proper ligation and care in dressing of the cord will in most cases prevent subsequent infection. Cleanliness is of the first importance. The hands, instruments, and tape used for ligation should be scrupulously clean. The best dressing for the cord is sterilized absorbent gauze several layers thick, and perforated in the centre. The cord is passed through this perforation and enclosed in the gauze. This dressing is renewed daily after the bath. The bath water must be clean, and in drying the infant care should be taken not to displace the cord. If a suppurating surface appears, it should be treated on general surgical principles. As a rule, ointments should be avoided. The ordinary sterilized wet dressing is sufficient.

### **Umbilical Fungus.**

(*Granuloma.*)

In some cases the stump does not heal after the cord has separated, and a granulating surface which presents a fungoid appearance remains. The granulating mass may become as large as a bean and be pedunculated. There is secretion of pus. The affection is a benign one, and should not be confounded with the so-called enteratomata, which are rare. The latter are composed of smooth muscular fibre and tubular glands. These umbilical tumors have been described by Kolaczek, who believes that they are formed by prolapsus of a persistent omphalomesenteric duct. Von Heukelom asserts that they are intestinal protrusions through true diverticula of Meckel. Adenoid tumors of the umbilicus have been described by Lannelongue and Fremont. Hüttenbrenner has reported a polypoid tumor of the umbilicus, which he believed to be the remains of the allantois.

**Treatment.**—If small and flat, the granulations are touched daily with silver nitrate and a dry dressing is applied; or the granulations may be carefully scraped off and the stump dressed with sterilized gauze after bleeding has ceased. If the growth is large and pedunculated, it should be ligated at its base with silk or catgut, and a sterile gauze dressing applied. In a day or two the mass separates and healing takes place.

### **Infection of the Umbilical Vessels; Arteritis Umbilicalis.**

In this affection the perivascular connective tissue of the cord first becomes infiltrated with serum and œdematous; later the various

coats of the arteries are affected. Thrombosis results, with disintegration of the thrombi. The lymph-vessels in the connective tissue of the cord carry the infectious material to the various parts of the body.

**Morbid Anatomy.**—The vessels running from the umbilicus appear as thickened discolored cords. The perivascular tissue is infiltrated. The process may begin about a centimetre behind the umbilicus and extend downward toward the bladder. The umbilical stump may be normal in appearance or inflamed. The lumen of the arteries contain thrombi. The vessels may be dilated and contain disintegrated purulent masses. There may be lobar or lobular pneumonia, with pleurisy and hemorrhagic infarction of the lung. Parenchymatous inflammation of the liver, kidney, and spleen, and suppuration of one or several joints (see Osteomyelitis) may be observed. Peritonitis may be a complication.

The bacteria found in most of these cases have been streptococci or staphylococci.

Umbilical arteritis is a wound infection. It is most frequently seen in institutions, and is the result of implantation of septic matter on the umbilical wound by the hands or instruments, or through the bath water or unclean dressings. Cases have occurred coincident with the presence of blennorrhœa.

The **symptoms** of arteritis umbilicalis are often indefinite and give no clue to the cause of the illness. The infants gradually emaciate and succumb, the fatal issue supervening quite suddenly. The umbilicus may in these cases have been long healed, and show no evidence of disease; in other cases it is inflamed. There is a sinus leading downward and backward toward the bladder, and from this pus exudes. A tense, cord-like structure, the inflamed umbilical vessels, is felt beneath the abdominal wall. Sometimes the first intimation of serious disease is seen in the joints. The mother may tell the physician that the infant cries when it is bathed or dressed. In these cases the knee, ankle, or hip may be swollen, tense, and the seat of exudate. A septic osteomyelitis of the epiphyses of the joint is present, resulting in a suppurative arthritis. As a rule, more than one joint is involved. In other cases the symptoms are indefinite; there is a slight febrile movement; the respirations are increased, and there may be a disturbance of the bowels; icterus is present. Physical examination may reveal a pneumonia or nephritis. In other words, the symptoms are those of sepsis.

**Prognosis.**—These cases are generally fatal. A few of the mild cases recover. In these, however, it is a question as to whether the vessels have been involved or whether there was a true infection of a septic nature. The prognosis is especially unfavorable in premature infants.



### Phlebitis Umbilicalis.

In this affection, the veins running from the umbilicus to the liver are the seat of an inflammatory process similar to that affecting the arteries in the affection just described. There is a true phlebitis, with pus in the veins, in some cases extending into the liver. The branches of the portal vein are involved. In these cases the symptoms resemble those of peritonitis complicated with icterus. The respirations are shallow, the abdomen tense, and the thighs are flexed on the abdomen.

**Treatment.**—It is hardly necessary to remind the student that prophylaxis is in all septic affections the mainstay of the physician. Once inaugurated, infective processes in newborn infants are progressive. In cases of the palpably umbilical type I have advised laying open the structures passing from the umbilicus to the bladder, curetting the sinus thus formed, and inducing healing from the bottom. Recovery has followed in a few exceptional cases. The operation should be performed before general infection has occurred. Van Arsdale operated on one of these cases for me, and obtained an apparent recovery—that is to say, the sinus leading from the umbilicus healed and there were no symptoms for weeks after the operation.

### Hemorrhage from the Umbilicus.

(*Omphalorrhagia.*)

Hemorrhage from the umbilicus may occur (*a*) from the vessels of the umbilical cord or (*b*) from the umbilical wound itself (parenchymatous).

Hemorrhage from the vessels of the cord may occur if the ligature has not been properly applied; but faulty ligation alone will not in all cases account for the hemorrhage. Runge states that if the cord is cut ten or fifteen minutes after a healthy infant has cried lustily, there will be little hemorrhage—certainly not one threatening life. The diminution of arterial pressure in the bloodvessels at this point, due to the establishment of the pulmonic circulation and the natural contractility of the vessels, will prevent hemorrhage. The fact that infants among savage peoples and the young of lower animals do not die although the cord is not ligated, but simply divided, is thus explained. If an infant, therefore, bleeds from an imperfectly applied ligature the reason must be sought in some physiological or anatomical defect of the bloodvessels. We possess no data to explain the absence of normal arterial contraction in the vessels of the cord. Inasmuch as this condition may be present during the first days after birth, great care should be taken that the ligature is properly placed. Caution should especially be exercised with premature infants, in whom the bloodvessels



are in an embryonal state. The method of ligating the cord will be found detailed in works on obstetrics.

After the separation of the umbilical stump a few drops of blood may be seen on the wound from time to time. This is of no moment. The wound should be dressed with a salicylic powder and amylum (1 : 5), and covered with a dry dressing.

### Idiopathic Hemorrhage from the Umbilicus.

(*True Omphalorrhagia.*)

**Occurrence.**—Winckel, quoted by Runge, has seen in 5000 births only 1 case of true idiopathic hemorrhage from the umbilicus. Males are more frequently attacked than females. I have seen few cases of this affection.

**Etiology.**—According to Grandidier, infants apparently healthy and strong are for the most part affected. This form of hemorrhage occurs also in infants suffering from congenital syphilis, septic affections, or the acute fatty degenerations of the newborn. In some forms of congenital syphilis there may be hemorrhages into the skin, stomach, intestine, and internal organs. In these cases it is not surprising that hemorrhage should also occur from the umbilicus. Icterus, due to syphilitic affections of the liver and lung, may be present.

In 51 cases of hemorrhage from the umbilicus, Epstein found pronounced septicæmia in 24. The affection is especially prevalent under unhygienic conditions and in foundling asylums. Klebs, Eppinger, Cohnheim, and Weigert have described cases of hemorrhage in which micro-organisms of various kinds were found in the blood and in the hemorrhagic areas. Bacterial colonies were found in the arterial thrombi and in the lungs and kidneys.

The occurrence of hemorrhage from the umbilicus in Buhl's disease is elsewhere referred to.

**Symptoms.**—About the fifth day after birth, immediately following separation of the umbilical stump, blood is seen to ooze from the umbilicus. It does not appear to issue from any particular vessel, but oozes from the whole wound, as from a sponge. The flow may be slight at first and then profuse, or may be profuse from the outset. Pressure upon the wound may cause the hemorrhage to cease, but the flow begins when pressure is withdrawn. In some cases the infants have enjoyed excellent health previous to the hemorrhage. In others there may have been a slight icterus or diarrhœa. However this may be, after bleeding commences cyanosis and icterus of the general surface appear, giving the skin a peculiarly bronzed appearance. There are hemorrhages from the stomach and gut. Ecchymoses appear in the vicinity of the umbilicus and on other parts of the trunk. Oedema of the ankle-joints and wrists supervenes.

The hemorrhage from the umbilicus is the most characteristic symptom, and cannot be controlled by any means. The blood coagulates very slowly.

**Duration.**—The disease lasts from a few hours to two weeks. Grandidier's statistics give a mortality of 83 per cent. Death ensues in collapse, with coma and convulsions.

**Treatment** is directed to controlling the hemorrhage by pressure or by transfixing the umbilical wound. From a study of the pathogeny of this affection, it is evident that no treatment can be successful.

### Umbilical Herniæ.

In newly born infants we distinguish two varieties of hernia at the umbilicus.

The first form is of serious character. It is really a hernia of the umbilical cord (*hernia funiculi umbilicalis*). The condition is due to an arrest of development, as a result of which there is a true defect in the abdominal wall at the situation of the umbilicus. The gut prolapses and is covered by the amnion of the cord and Wharton's jelly, beneath which is the peritoneum. The latter is immediately over the gut. Many of the infants thus affected are premature. In others deformities are present. The hernia is a round or oval tumor of the size of a walnut or an orange, located in the region of the umbilicus, and is continuous with the cord. The sac of the hernia is formed by the peritoneum and amnion. The abdominal walls form the border of the sac. Gut, liver, spleen, kidney, or pancreas may be found in the sac.

If treatment is not instituted at the time of separation of the cord, and the hernia is large, ulceration, gangrene, or septic peritonitis in the sac contents may result.

The second and most common form of hernia in this region is due to a weakness at the point of insertion of the cord. The hernia becomes apparent a few weeks after birth, when the cord has completely cicatrized. It is then noticed that when the infant cries there is a protrusion at this point. The protrusion may be small or large, and is covered by the thin cicatrized skin. The hernia may be central or at one side, or a little above or below the centre of the umbilical ring.

The **treatment** of the first form is purely surgical, and consists in splitting open the sac and sewing the abdominal parietes in apposition. The treatment of the second form is simple. As a prophylactic measure a small pad should be placed on the abdomen, underneath the binder, and should be worn for some time after the stump is healed, in order that there may be no protrusion of the wall and gut during crying spells. If the hernia has taken place, a firm pad, made by enclosing a piece of thick cardboard, one



and a half inches in diameter, in a piece of linen, should be applied, and supported by rubber plaster. Another method is to reduce the hernia, fold it inward by means of the apposing abdominal walls, and secure the walls thus brought together with plaster. The plaster should be renewed every three days lest ulceration of the skin result. As soon as the muscles of the abdomen gain strength and the infant is able to stand, the opening at the umbilicus closes and the hernia remains reduced.

### MELÆNA NEONATORUM.

This is a disease of the newly born characterized by a discharge of blood from the rectum and by vomiting of blood. It is a rare affection, occurring about once in 1000 births (Kling, Genrich, Runge). The hemorrhages occur in two distinct conditions:

(a) As a symptom of a constitutional dyscrasia. This condition has been treated of under the headings of Hemorrhagic Congenital Syphilis, Sepsis, and the Acute Fatty Degeneration of the Newly Born. Runge has shown that not only may these diseases named cause melæna, but also that any of the infectious diseases of the newly born may give rise to this condition.

(b) The second condition in which melæna occurs is that in which, as Landau, in his monograph on this disease has shown, local lesions, such as erosions and ulcerations resembling *ulcus ventriculi*, exist in the stomach and gut of the newly born infant. Hecker, Spiegelberg, and others have also described these ulcers of the stomach which produce the symptoms of melæna. Landau attributes the ulcer to embolism resulting from a thrombus of the umbilical vein or the ductus Botalli. Embolism in any artery of the mucous membrane of the stomach gives rise to necrosis and erosion, with the opening up of some arterial branch. Ingenious as this theory is, it is not accepted unreservedly by all (Kundrat), although Landau has proved the presence of emboli in the vicinity of stomach ulcerations. Another theory ascribes the ulcerations to hyperæmia of the mucous membrane in asphyxia and traumatism.

In addition there are cases in which no cause for the symptoms can be detected.

**Morbid Anatomy.**—Post-mortem examination shows the gastro-enteric tract to be filled with dark hemorrhagic masses. The mucous membrane may be normal, the seat of erosions of greater or lesser extent, or there may be hemorrhagic areas scattered throughout the gut. These may be confined to the stomach or duodenum. There may be true ulcers, measuring  $\frac{1}{2}$  to 2 cm. in diameter, resembling those seen in the adult (Winckel). In some cases the thrombosed or eroded vessel is found in the floor of the



ulcer or in its vicinity. All the organs are anæmic, and if syphilis or some other general disease exists we have the changes found in these conditions.

**Symptoms.**—From two to four days after birth it is noticed that the infant is somnolent or restless; there may be hemorrhagic stools or vomiting of bloody masses, or both these symptoms may be present at the same time. The principal symptom, however, is the bloody stools. These are at first mingled with meconium, and later become frequent and profuse. The vomited matter consists of mucus streaked with blood, or masses of blood of brownish color. The amount of blood lost by the bowel within twenty-four hours may be enormous. Under these conditions death ensues within a period of from twelve to twenty-four hours, with all the symptoms of acute anæmia. In other cases there may be a cessation of the intestinal hemorrhage for from twenty-four to forty-eight hours, but recovery does not always take place, and sudden death from a severe hemorrhage may occur at any time.

The **prognosis** is grave. Sixty per cent. of the infants affected die. The outlook is more serious in conditions of sepsis, syphilis, and acute fatty degeneration than in melæna due to ulcer of the stomach or duodenum.

**Diagnosis.**—We must differentiate this disease, which is called true melæna, from the so-called spurious forms, in which the infant simply passes blood swallowed with the food. This spurious form may occur if the breast nipple is fissured or if there is a fissure of the anus. In other cases blood from the nose or mouth of the infant may be swallowed. Hemorrhages of this kind may occur as part of a general septic infection. In many cases there may be, with other hemorrhages, icterus, cyanosis, œdema, pointing to some general disease. Sensitiveness in the region of the stomach points to ulceration of this organ.

**Treatment.**—The hemorrhages should be controlled by the application of a cold coil to the epigastrium and the administration of cold drinks. Hænoch recommends a drop of liquor ferri sesquichloridi every hour in barley-water. Ergotin is given in doses of  $\frac{1}{2}$  to  $\frac{3}{4}$  grain internally or subcutaneously. Enemata are not advisable. Heart is stimulated with strychnine, digitalis, camphor, or ether.

### ACUTE FATTY DEGENERATION OF THE NEWBORN INFANT.

(*Buhl's Disease.*)

This disease, first described in 1861 by Buhl, is an acute parenchymatous fatty degeneration of the liver, kidney, or heart, combined with hemorrhages into the various organs, or from the umbilicus, intestines, or stomach.

**Etiology.**—The disease is found in the lower animals, especially in sheep. In the human subject it is probably a form of septic infection, although in Buhl's cases the vessels of the umbilicus had a normal appearance. Runge believes that with modern histological and bacteriological methods an infectious agent will be ultimately discovered. Septic infection may occur without any appreciable changes about the umbilicus or elsewhere on the surface of the body (cryptogenetic). The disease is very rare; many cases described as omphalitis and hemorrhage from the umbilicus are probably Buhl's disease.

**Morbid Anatomy.**—The body is icteric or cyanotic; there is œdema of the surface, and not infrequently hemorrhagic areas in the skin. The umbilicus may be covered with blood, but the vessels and wound are otherwise normal. Hemorrhages or petechiæ are found in most of the internal organs, especially the pleura, pericardium, mediastinal tissue, muscles, and mucous membranes. The heart is the seat of fatty degeneration, as is also the liver which is enlarged. The spleen is enlarged and soft. The kidneys are the seat of fatty parenchymatous changes. The stomach and intestines are filled with blood. There are hemorrhages into the mucous membrane of the stomach and intestine.

**Symptoms.**—The children are born partially asphyxiated. Attempts to resuscitate them are not fully successful. Some die in asphyxia, others after a time have bloody diarrhœal stools. At times there is vomiting of blood, and when the stump of the cord separates there is hemorrhage from the umbilicus. The bleeding from the umbilicus is parenchymatous, and may be so profuse as to cause death. The skin is at first cyanotic, then icteric in hue. Large hemorrhagic areas appear in the skin, conjunctivæ, and mucous membrane of the mouth, and bleeding may occur from the ear and nose. Icterus may become extreme. At times œdema of the surface appears. The temperature is not raised. Death ensues in collapse. The external hemorrhages and icterus are absent in some cases.

**Diagnosis.**—In the newly born infant this symptom-complex is unique, and must be looked upon as a form of sepsis either through the umbilicus or through some other avenue. In the newly born infant this disease may be confounded with death from asphyxia. In all cases of medico-legal import the organs should be examined for parenchymatous changes before an opinion is given.

**Prognosis** is fatal.

**Treatment.**—The physician endeavors to bring the infant out of the state of asphyxia. It can be easily understood that he stands helpless in the face of the parenchymatous hemorrhages and degenerations, for which there is no remedy.



**WINCKEL'S DISEASE.**

(*Epidemic Hæmoglobinuria of the Newly Born.*)

This disease, first described in the epidemic form by Winckel, is characterized by the sudden appearance of cyanosis and icterus with hæmoglobinuria.

The **etiology** of the affection is obscure. The symptomatology resembles that of Buhl's disease. Epstein, Strelitz, and Baginsky consider the disease a form of septic infection. Winckel's cases were believed to be due to the use of infected drinking or bath water.

**Symptoms.**—The symptoms in Winckel's cases appeared on the fourth day after birth in apparently healthy and well-developed newly born infants. The average duration was thirty-two hours. Some infants succumbed in nine hours after the onset of symptoms. Restlessness and cyanosis were first noted. The latter was general, affecting the trunk and extremities. Icterus then developed, and became marked within twenty-four hours. The respiration and pulse were increased; the temperature was normal (100.5° F., 38° C.); the skin was cool. At times there were vomiting and diarrhœa. The urine was passed with tenesmus, was brownish in color, and contained blood-cells, hæmoglobin, renal epithelium, granular casts, micrococci, detritus, and ammonium urate. Convulsions closed the scene. If the skin was cut, a brownish syrupy fluid escaped.

Post-mortem examination revealed no disease of the umbilicus or umbilical vessels. The kidneys were the seat of cortical hemorrhages. The spleen was large and hard, and filled with pigment. There were punctate hemorrhages in almost all the organs, especially in the pleura, pericardium, and endocardium. Hemorrhages were present in the mucous membrane of the stomach and gut, and underneath the liver capsule. Peyer's patches, solitary follicles, and mesenteric glands were enlarged. The liver, heart, and various organs showed fatty degeneration. There were bacterial foci in the liver and kidneys. The blood showed an increase in the leucocytes and in the free granules.

**Diagnosis.**—Owing to the similarity of symptoms, Winckel's disease may be confounded with Buhl's disease. The former pursues a very malignant course, and does not present the intestinal and stomach hemorrhages to the same extent as the latter.

Runge and others are inclined to believe that all these hemorrhagic affections are due to a common cause—septic infection. The hæmoglobinuria is simply a marked hemorrhage into the kidney. Parenchymatous fatty degeneration of the various organs is common to both affections.



**TETANUS OF THE NEWBORN INFANT.**

(*Trismus Neonatorum.*)

Tetanus of the newborn infant is in the majority of cases due to infection of the umbilical wound by the tetanus bacillus. The bacillus is conveyed to the wound by means of unclean hands, bandages, or filth of any kind. As a result of the growth of the bacillus ptomaines (Brieger) are formed, enter the circulation and are widely distributed throughout the body. Infection may occur at the time of the ligation of the cord, or during the separation of the stump. In 8 per cent. of the cases the disease manifests itself immediately after birth (Hartigan). As a rule, however, it occurs from the fifth to the twelfth day after birth (Runge). It is rare after the third week. It is common in districts in which uncleanness in the methods of treating the umbilical cord prevails. It is endemic in the Faroe islands, and is common in the Hebrides, Cuba, and Jamaica. Negroes especially are prone to the malady, on account of their lack of cleanliness in treating the cord. Tetanus of the newborn infant has been demonstrated by Beumer and Peiper to be identical with tetanus in the adult.

**Morbid Anatomy.**—Beck has described two cases of tetanus with swelling of the motor ganglion-cell, and degeneration of the peripheral portion of the cell and atrophy. There are also changes in the chromatin. Congestion and hemorrhages in the brain and cord, serous exudates in the cord, and congestion of the internal organs, due to convulsions, are present.

**Symptoms.**—There is a premonitory period of restlessness. The infants awake abruptly from sleep. They nurse badly, let go of the nipple suddenly, and cry. The peculiarity of the disease in infants is the predominance of trismus, with which the attack begins. The lower jaw becomes rigid and fixed at a distance of a few lines from the upper jaw. It is impossible to introduce the nipple between the teeth. The forehead is wrinkled, the palpebral fissure diminished, and the lips are puckered. At intervals this spasm relaxes. The condition of rigid spasm spreads to the muscles of the body, and there is opisthotonos. At the outset during the intervals between the attacks of rigidity the body is lax; these intervals become shorter and shorter, until finally the body is in a state of constant rigidity, resting on the heels and the back of the head. Dyspnœa with resultant cyanosis is present when the muscles of respiration become affected. Deglutition is impossible. There is no cry, on account of spasm of the laryngeal muscles. The temperature may reach 106° F. (41° C.). In protracted cases it may be normal. The urine and feces are passed involuntarily. There is albumin in the urine. The respirations are superficial. The heart

action is increased; the pulse may be 200. During a contracture the skin is dark red and cyanotic. Icterus may be present. The face is fixed in expression and oedematous.

**Duration.**—The disease lasts from a few days to three weeks. Death may ensue in from one to six days from asphyxia or exhaustion. In rare cases the attacks become less and less frequent, and finally cease. Fracture of the bones and rupture of the muscles are among the complications.

**Prognosis.**—The prognosis is grave. Baginsky lost all of his cases in newborn infants, while Escherich, Soltman, and Monti report recoveries. Cases which occur late, after separation of the cord, give a better prognosis (Papiewski).

**Treatment.**—Prophylaxis is of the utmost importance in this, as in other diseases of the newborn infant. Cleanliness in handling the cord is of the first importance. Escherich cauterizes the stump of the cord, to destroy any bacilli of tetanus which may be present. On the appearance of trismus, the treatment is first directed to the relief of the tonic spasms. Chloral hydrate in grain  $j$  (0.06) doses every few hours, by mouth (if possible), or by the rectum, is a very useful drug. Calabar bean in the form of extract is recommended by Monti, who gives  $\frac{1}{120}$  grain (0.0005) subcutaneously, repeated until the desired effect is obtained. Cannabis indica, grain  $\frac{1}{2}$  (0.03) every two hours, is also given internally. Curare has been used but little with the newborn infant. Of the other remedies, bromide of potassium and trionol have little effect.

The use of the tetanus antitoxins has not given satisfactory results, probably owing to the fact that tetanus is a symptom of advanced toxæmia of the nervous system. In such a condition the action of any antitoxin would be exerted too late to give permanent benefit.

### OPHTHALMIA NEONATORUM.

Ophthalmia neonatorum is an inflammation of the conjunctivæ, of gonorrhœal origin. The infant may be infected intrapartum or after birth. The source of the infection may be the parturient canal of the mother, or the infectious agent may be conveyed to the eyes by the finger of the accoucheur. Postpartum the infant may be infected by any means which conveys gonococci to the orbital conjunctivæ. In institutions an infant may be infected by a careless nurse's washing the eyes with unclean linen. The period which elapses from the time of infection to the onset of symptoms varies from two to five days. There is a discharge from the conjunctivæ, the eyes are congested, the conjunctivæ swollen, and the eyelids swollen and chemosed. At first a thin yellow serous discharge escapes from between the lids. This soon becomes thick and



creamy and of a yellowish or icteric hue if icterus is present. The lids are so swollen and cedematous that it is scarcely possible to separate them. When separated the conjunctiva of the lids prolapses. The cornea is rough and covered with secretion, and shreds of pseudomembrane may adhere to the palpebral conjunctiva. If not controlled, the inflammation progresses until the whole depth of the cornea is involved and perforation occurs, with prolapse of the iris, escape of the humor, and panophthalmitis.

The duration of the disease varies ; as a rule, it lasts from three to five weeks.

This gonorrhœal form of ophthalmia must not be confounded with a much milder form of conjunctivitis which is also seen in the newly born infant. In these cases, also, the infant is infected by the mother or nurse, but the inflammation is of a benign nature, and is not due to the gonococcus of Neisser. In this form of ophthalmia the diplococci and streptococci of the vagina of the mother are the etiological factors. The swelling of the lids is not marked, the symptoms are mild, and the course favorable.

**Prognosis.**—The prognosis of gonorrhœal ophthalmia depends on an early diagnosis, the degree of the severity of the infection, and timely treatment. It is grave in all cases.

**Treatment.**—The advice of an ophthalmic surgeon should be sought as soon as symptoms are manifest. As a prophylactic measure, immediately after birth a drop of a 2 per cent. solution of silver nitrate should be instilled into the eyes. The immediate result is a cloudiness of the cornea, due to the formation of albuminate of silver. The cloudiness disappears in a few days. If only one eye is affected, the healthy eye should be carefully bandaged, so that infection cannot reach it. The infant should be placed in the hands of a special nurse, and pieces of lint, two inches square, should be kept on ice and applied to the lids every ten minutes. The secretion should be washed away as soon as formed, with a 1 : 500 boric acid solution applied with a dropper. Some surgeons wash the eyelids and conjunctivæ daily with a 1 to 3 per cent. solution of silver nitrate. The infant and nurse should be isolated, and all dressings burned as soon as soiled.

### ICTERUS IN THE NEWBORN INFANT.

The majority of newly born infants are icteric. Icterus in the otherwise normal newly born infant should be differentiated from that due to sepsis, syphilis of the liver, cirrhosis of the liver, stenosis of the common bile-duct, and yellow atrophy of the liver. Acute yellow atrophy of the liver in the mother during pregnancy may produce an icteric condition in the newborn infant.



### Icterus Neonatorum.

Opportunity to inspect post mortem the viscera of cases of icterus neonatorum is rarely afforded, since recovery ensues in the majority of cases. In cases which have come to the autopsy table, all the internal organs, including the bones and cartilages, were icteric. The spleen and kidneys were but little affected, even in severe forms, by the general icteric discoloration. The liver was macroscopically rarely jaundiced. The intima of the arteries, the fluids in the serous cavities, the pericardial fluid, and the subcutaneous and intermuscular connective tissue have been found to contain bile-pigment and biliary acids (Birch-Hirschfeld). The contents of the gut were normal. The kidneys contained uric acid infarctions.

**Symptoms.**—Fully 80 per cent. of all newly born infants become jaundiced shortly after birth (Runge). The jaundice appears on the second or third day after birth. The icterus may be slight and involve only the face, breast, and back, or may be severe and be seen over the whole surface. In severe forms icterus of the conjunctivæ is present. In this feature icterus neonatorum differs from ordinary catarrhal icterus, in which icterus of the conjunctivæ is the first symptom before the skin is perceptibly tinged. The conjunctivæ are last to be tinged in the jaundice of the newly born. Infants suffering from icterus, though in an apparently normal condition, do not increase in weight as normal infants do. They may even lose ground. When they recover lost weight, they do so slowly.

The urine is brownish at times and contains biliary pigment and acids (Cruse, Hofmeier).

**Etiology.**—Icterus neonatorum is as frequent in institutions as in private practice. It is more common among boys (Kehrer). It is seen in premature weakly infants, and in those whose birth has been attended by complications. The disease is now traced to both a hæmatogenous and a hepatogenous source. There are certain processes in the blood which also involve the functions of the liver. According to Hofmeier and Silbermann, there is a disintegration of red blood-cells in the circulation. These disintegrated red blood-cells are converted by the liver cell into biliary pigment; the solids of the bile are increased, as is also the gross quantity of bile (Minkowski, Naunyn, Stadelmann). It is not known, however, how this increase of bile-pigment gains access to the circulation. One theory (Silbermann) is that with the processes described above, certain ferments are set free which cause circulatory disturbances in the liver. Stasis results in the bloodvessels, with consequent pressure on the biliary ducts. Resorption of bile thus follows.

**Treatment.**—Icterus neonatorum, if untreated, disappears in three or four days in mild cases; severe cases are more protracted. Neither form needs special treatment.

**SCLEREMA.**

(*Sclerema Neonatorum* ; *Sclerœdema* ; *Sclerema Adiposum*.)

This peculiar and rare affection is apt to be confounded with ordinary œdema. There are two forms of this condition : sclerœdema, or œdematous sclerema ; sclerema adiposum, or fat sclerema.

**Sclerœdema.**

Sclerœdema is an accumulation of fluid in the subcutaneous connective tissue, causing the skin to be raised from that tissue. The skin resembles marble, and is hard, tense, and glistening. The infiltration causes a stiffness of the body, with rigidity of the extremities. The swelling begins, as a rule, in the calves of the legs, and spreads to the buttocks, thighs, and trunk. The nose, cheeks, and lips become hard, and the skin is lardaceous. The joints are stiff ; the infant does not nurse, being unable to suckle. The surface is cool, and the internal temperature may fall to 89.6° F. (32° C.) or 71.5° F. (22° C.). There is in this form a true exudate underneath the skin. In some cases we may have ecchymoses in the tense, white, glistening skin. It affects weakly newborn infants.

**Sclerema Adiposum, or Fat Sclerema.**

This condition follows or complicates exhausting diseases, and is also seen complicating diarrhœa or pneumonia. It is generally a forerunner of death. The skin becomes hard, but is not lifted up by an exudate, and the limbs are immovable. On the contrary, the skin is collapsed, and resembles that seen in atrophic states. This condition is really a dryness of the tissues. The skin has lost its natural resiliency, but does not pit on pressure. The hardening begins on the lower extremities, and the inner part of the thighs or in the cheeks. It spreads thence. The skin is not tense. The external and internal temperatures are reduced. The condition ends usually like the first form, fatally. It is not confined to newborn infants, but may affect older ones. In these cases also the sclerema affects the whole trunk and may spread to the face. The cheeks cannot be lifted up in folds.

**Diagnosis.**—The diagnosis is not difficult. The condition in which the skin is tense and does not pit as in œdema is characteristic. In the second form the skin feels much like that of a corpse. It may even retain its original wrinkled condition if the infant is atrophic. With our present imperfect knowledge it is well to keep these forms of disease in a class apart from the sclerema with sclerodactylia seen in adults or in older children.



The **etiology** of sclerœdema or acute œdema is still a matter of speculation. Weakness of the heart, the beginning of nephritis, an infectious agent of some kind (Baginsky), certain marked deficiencies in the respiration and circulation in premature infants, and unhygienic surroundings, all have been advanced to explain this rare condition. In the secondary form of sclerema adiposum there is to a certain extent a desiccation of the subcutaneous tissues. Sânger thinks that the excess of palmitin and stearin in the subcutaneous fat of the newly born infant may account for the peculiar solidification as soon as the temperature is reduced, as it is in sclerema. There are cases of sclerema in which the temperature is elevated, as in Barker's case, so that the theory of Sânger is scarcely adequate. The cases of fat sclerema which I have seen created the impression of an infectious condition. Barker found streptococci in the internal fluids after death.

**Prognosis.**—Most infants having the œdematous form die. I have seen one case of fat sclerema involving the buttocks and inner part of the thighs, recover.

**Treatment.**—With a view to prophylaxis, the utmost cleanliness should be observed at birth, and extreme care taken with the cord and its dressing. If the temperature is subnormal or the infant premature, every effort should be made to supply the requisite warmth by artificial means. The nourishment should be carefully selected. In the secondary form of sclerema the same general treatment is pursued. The heart is stimulated and heat is supplied artificially. Localized forms are treated with cautious massage of the affected areas.

## INJURIES INFLICTED DURING BIRTH.

Among the injuries incident to birth are those of the face. Pressure of the forceps blade may cause facial paralysis. This, as a rule, disappears in time, though in severe injury of the nerves it may remain permanent. Indentations of the cranial bones may result from the pressure of instruments. In these cases the bone is depressed, and in the space between the scalp and bone there is an effusion. The edge of the bone surrounding the depression is distinctly felt. These depressions need no treatment, as they disappear in time. Traction on the arm may cause a so-called birth palsy, which is the counterpart of Erb's palsy in later life. The paralysis in these cases sometimes remains permanent. Others recover. As a rule, one arm is affected, but in rare cases both arms may be paralyzed. The symptoms are characteristic. In a few days or at a later period after birth it is noticed that the infant does not move one or the other arm (Fig. 27). The affected limb hangs

loosely and without power of motion. The fingers or hands may be mobile. The affected arm is cold and the hand may be bluish in tint. After a time atrophy of the muscles about the shoulder-joint may set in. The bony prominences then come into relief. If the arm does not recover power, the muscles continue to atrophy, and there may be subluxation of the head of the humerus at the shoulder-joint. The child in these cases always holds the injured arm with the sound one, in order to protect and support it. At the early period the reactions of degeneration are present, and if the muscles recover, the reaction to the galvanic and faradic current

FIG. 27.



Birth palsy affecting the left arm, atrophy of the muscles about the shoulder.

becomes normal. If recovery does not take place, the disappearance of galvanic and faradic irritability of muscle goes hand in hand with the muscular atrophy.

The **treatment** of these obstetrical palsies is similar to that of Erb's palsy. The arm is protected from traumatism. Massage is performed within two weeks after injury, and after four weeks the faradic current is applied to cause muscular contraction. Electricity is applied for a short space of time daily. The progress of these cases can best be judged under treatment. As a rule, recovery takes place in a few weeks. In other cases recovery may be delayed. In a third set of cases recovery never takes place. The galvanic and faradic contractility disappears from muscle and nerve, and permanent atrophy and disability remain. In these cases there is also retarded growth of the other tissues, such as bone.





PLATE VI.



Hæmatoma of the Sternomastoid Muscle of the Right Side in a Newborn Infant. Swelling at the centre of the anterior border of the muscle; contraction of the muscle with torticollis.



### Hæmatoma of the Sternomastoid Muscle.

This affection is the direct result of traumatism during delivery. As a rule, it is seen in cases of breech presentation in which traction has been exerted on the after-coming head. In the majority of the cases coming under my observation the sternomastoid muscle of the right side was affected (Plate VI.). The infant holds the head on one side. The muscle of the affected side is contracted, and the position of the head is that seen in torticollis. A hard nodule is felt along the inner border of the sternomastoid muscle, about the junction of the lower third and upper two-thirds. The tumor is usually the size of a small hazelnut, but may be much larger. Manipulation causes pain. The skin over the tumor is movable and not discolored.

The progress of the affection in all of these cases is much the same. The tumor becomes smaller as the exudate is absorbed, but the torticollis persists, although in time this may disappear. The nature of these tumors is probably that of a hæmatoma caused by rupture of muscular fibres and bloodvessels.

The **treatment** is simple. At first the tumor should be let alone. After a few days gentle massage with the finger moistened with oil is permissible. When the growth hardens the massage may be more vigorous, and be supplemented with an attempt at each sitting to turn the head gently to the opposite side and thus stretch the contracted muscle. Cases which do not recover must be treated by surgical means later in life.

### Cephalohæmatoma.

Cephalohæmatoma is an effusion of blood between the pericranium and the skull-cap. The pericranium and scalp are raised into a distinct tumor. In external cephalohæmatoma the effusion is between the pericranium and the skull; in internal cephalohæmatoma it is between the dura mater and the skull. Kee found both forms present in the same patient in 9 out of 20 cases.

**Symptoms.**—There is a tumor varying in size from that of a hazel-nut to that of an orange, of elastic consistency, situated in most cases on one or the other parietal bone. It is round, elongated, or kidney-shaped. It covers part or the whole of the bone, but never extends beyond the sutures. The skin over the tumor is not sensitive to the touch, is normal or slightly bluish in color, and is perfectly movable over the tumor. After a few days the circumference of the tumor is bounded by a distinct wall, at first soft, but later of bony hardness. The general health of the infant remains good unless there is a complication. This blood tumor appears two or three days after birth. At first it is tense, but afterward becomes softer

and doughy to the touch. It reaches its maximum size in from six to eight days. It begins to diminish in the second week, and disappears by the fifteenth week. The tumor is either absorbed or there is a proliferation of bone, which remains as an exostosis. At this time crepitation resembling that of parchment is felt. Around the former tumor a thin wall of bone is found.

**Occurrence.**—These tumors are not common. Hennig found 230 cases in 53,506 births, or 0.43 per cent. of the whole number. Hofmohl's statistics give a like figure. Most of the cases are vertex presentations. The cephalohæmatoma usually occurs on the right parietal bone, and may follow easy as well as difficult labors. It is present oftener in boys than in girls, and is seen in premature infants as well as full-term babies. It has been observed in breech cases, especially if forceps has been applied to the after-coming head. These tumors may occur on both parietal bones of the infant. In such cases the sagittal suture distinctly separates the two tumors.

**Complications.**—Internal cephalohæmatoma, or cerebral hemorrhage, may complicate the external tumor. In such cases there has been a difficult labor with the application of forceps. The majority of the infants thus affected die. Suppuration of the tumor may take place, or diffuse cranial phlegmon may result fatally. A section of a cephalohæmatoma shows the scalp to be studded with punctate hemorrhages. The pericranium is bluish and covered with hemorrhages, and is separated from the skull by a collection of fluid blood under great tension. The bone beneath is rough or covered with a few clots. A bony wall is seen around the circumference of the tumor. It is a periosteal formation. After a time the bone and the inner surface of the pericranium become coated with a gelatinous exudate, which is subsequently converted into bone. In some cases quite an extensive bloody effusion is found between the dura and skull.

The situation of the cephalohæmatoma always corresponds to the position of certain natural fissures which exist in the posterior part of both parietal bones, running from the sagittal suture. In the occipital bone these fissures radiate from the lateral fontanelles and separate the upper and the inferior part of the occipital bone.

**Pathogenesis.**—A cephalohæmatoma is the result of the bursting of a small vessel between the periosteum and bone, and at the situation of the caput succedaneum. Hence the frequent formation of the tumor on the right parietal bone. It is most common in first-born infants. Asphyxia of the infant favors the formation of the tumor. Cephalohæmatoma may also occur as a part of the hemorrhagic symptomatology in general diseases, such as syphilis, sepsis, and Buhl's disease.

The **diagnosis** is made in the presence of an elastic fluctuating tumor distinctly limited by suture and surrounded by a ring or wall.



A caput succedaneum is œdematous and bluish, is seen immediately after birth, passes beyond the sutures, does not fluctuate, and disappears shortly after birth. A hernia of the brain does not fluctuate, grows tense when the infant cries, and shows respiratory fluctuations and pulsation. It can be reduced. Abscess of the scalp is painful, hot, and red; the phlegmon spreads over the whole scalp and is accompanied by œdema of the whole region. If cerebral symptoms are present with a cephalohæmatoma, they point to corresponding internal effusion or cerebral hemorrhage.

The **prognosis** is good if there is no internal tumor or cerebral hemorrhage, or if infection of the external tumor with resulting abscess does not occur. Even the latter, however, does not preclude the possibility of recovery. The prognosis is bad if the cephalohæmatoma is part of a general hemorrhagic condition, as in syphilis, fatty degeneration, or sepsis.

**Treatment.**—Uncomplicated cephalohæmatomata are absorbed if let alone. If abscess occurs, the tumor should be opened under antiseptic precautions, evacuated, and the sac packed with iodoform gauze.

On the other hand, even in the early stage, the tumor may be large and tense, and cerebral symptoms may be present. Such effusions of blood may communicate with an internal tumor through the parietal or occipital fissures mentioned. In such very exceptional cases aspiration to relieve internal pressure may be justifiable (Runge).

#### LEADING AUTHORITIES REFERRED TO IN CHAPTER II.

- Bertin, G.*: Infections des Nouveaux nés dans le couveuse, Paris, 1899.  
*Escherich, T.*: "Trismus et Tetanus," Wien. klin. Wochenschr., 1893. "Brutkammer für frühgeborenen," etc., Mittheil. Vereins Steiermark, No. 3, 1900.  
*Fischl, R.*: "Infection Septique," Traité Maladies des Enfants, Comby, 1896.  
 Prophylaxe der Krankheiten des Kindesalters, Nobling, Jankau, 1900.  
*Landau, L.*: Melaena der Neugeborenen, Breslau, 1874.  
*Moschowitz, A. V.*: "Tetanus," Annals of Surgery, 1900.  
*Pascaud, V.*: La Couveuse Artificielle, Paris, 1899.  
*Runge, M.*: Die Krankheiten der Ersten Lebensstage, 1893.  
*Voorhees, J. D.*: "Care of Premature Babies in Incubators," Arch. of Pediat., May, 1900.

## CHAPTER III.

### THE SPECIFIC INFECTIOUS DISEASES.

#### THE EXANTHEMATA.

THE exanthemata, scarlet fever, measles, R  theln, varicella, and variola, are acute specific infectious diseases. They form a distinct group. The poison or infectious element originates in the body of the patient. The nature of this poison is unknown. Though suspected to be bacterial, the essential cause in any of the exanthemata has not been isolated. We do know, however, that the acute exanthemata are conveyed from one person to another through the medium of the atmosphere. In this respect they differ essentially from such diseases as typhoid fever, or even syphilis, in which the morbid agent must be introduced into the body. They are therefore contagious in the true sense of the term. Most people are susceptible to some of the exanthemata, such as measles and smallpox. On the other hand, not every one exposed to contagion will contract scarlet fever or varicella. Few persons are attacked twice by the same exanthematic affection, but there are exceptions to this rule. An attack of one disease, such as measles, does not confer immunity from an attack of another, such as scarlet fever. The exanthemata occur either endemically or epidemically. Each has a well-defined period of incubation—that is to say, an interval between the time of the exposure to contagion and the onset of characteristic symptoms. In the different exanthemata this interval varies within wide limits. The period of incubation seems to be more accurately determined in measles than in the other exanthemata. It is well established that two of the exanthemata may occur at the same time in the same subject. This is not a point in favor of the identity of the essential cause of the exanthemata. On the contrary, it is an accepted fact that each of the exanthemata is distinct in itself, and that each disease has its specific essential cause. The exanthemata are characterized by an eruption on the skin, the so-called exanthema, or rash.

#### SCARLET FEVER.

Scarlet fever is an acute infectious disease with a characteristic rash or exanthema. It is highly contagious.

**Etiology.**—It has not as yet been established whether the infec-



tious agent is a micro-organism, although streptococci have been isolated from the secretions and scales in the desquamative period. Neither do we know whether there is an organism in the circulating blood. The atmosphere about the patient seems in most cases to be the zone of contagion. The nearer a person has been to the patient the more likely is he to convey the disease to a third person. Articles of clothing may retain the infection for months. Scales from the skin of the patient, dried secretions, the urine if nephritis exists, and feces are also mediums of infection. The longer the physician remains near the patient the more likely is he to convey the disease. This mode of infection occurs. Osler records his belief in having carried infection to a patient. Foodstuffs handled by those suffering from the disease or by those who have been near patients may convey the disease. This is especially the case with milk, which is said to have been the cause of epidemics in England. The poison of scarlet fever seems to pervade the ward or sick-room for a long time. Whether this period extends over two years, as recorded by Murchison, is a matter not yet settled. We do not yet know how the poison obtains entrance to the body. The discharge from a scarlatinal otitis is said to be capable of communicating the disease.

**Susceptibility.**—All children exposed to infection do not contract the disease. It is less contagious than measles. On the other hand, although a person may be exposed once and escape, he is not necessarily immune to future exposures. A nurse attended many cases for me before contracting the disease. As a rule, one attack of scarlet fever protects a person from subsequent attacks. The literature records cases of well-observed second and third attacks. The author has met cases of a second attack. We should, however, be cautious in accepting reports of repeated attacks. R $\ddot{o}$ theln may have been mistaken for scarlet fever.

**Occurrence.**—Scarlet fever occurs at any age, and in all countries, being endemic in North America and Europe. It is most prevalent in autumn and winter (September to February). It remains endemic wherever introduced. Sporadic cases occur. It occurs also in epidemics. In epidemics only 38 per cent. of the population are affected. There is therefore an immunity of the majority (J $\ddot{u}$ rgensen, on the Faroe Epidemics). As a rule, fully 56 per cent. of those exposed before the twentieth year contract the disease.

**Incubation.**—According to the German authorities, scarlet fever has an incubation period of from eight to eleven days. English authors (Murchison) fix the period at from three to six days. The vast majority of cases develop within a period of from three to five days after exposure. If eleven days elapse without the appearance of symptoms, we may with reasonable certainty say that

the danger is past. Cases of thirty days' incubation are recorded, and the author had a case in his practice in which a physician conveyed the disease, the boy being attacked three weeks after his visit. In all such prolonged periods of incubation, however, there is a probability of a more recent exposure. The contagion is active during the period of incubation and during the eruptive and desquamative stages. The consensus of opinion is that the contagion diminishes in the desquamative stage. We should exercise great caution in allowing convalescents to communicate with the healthy. Strange to say, there are no positive data on this point. Contagion will be treated more fully under Prophylaxis.

**Immunity.**—Although there is no absolute immunity at any age, scarlet fever attacks nursing infants less frequently than older children. We have no positive data as to transmission of the affection in utero. Cases are recorded in which the newly born infant has been attacked, but some authors are inclined to look on such cases with doubt. In certain sets of cases the affection takes on a virulent form—cases in families in which all the members attacked will have complications, septic or otherwise, of a fatal character. An instance came under the author's notice in which during a very ordinary epidemic of scarlet fever one family lost two of three children attacked. All had septic malignant fever. There may in such cases be an element of mixed infection (Hench).

**Symptomatology.**—Scarlet fever does not present uniform symptoms. A general description of the disease can hardly be given without misleading the student. During an epidemic or during the prevalence of scarlet fever, there are a number of cases of angina in which no exanthema of scarlet fever is seen. This is especially so with those whose duties keep them near scarlet fever patients. There is no doubt that such anginal cases are capable of conveying the disease to others. A case of this kind has come under the author's notice. A nurse suffering from an angina went from a scarlet fever case to a healthy child. Although the nurse had taken all external precautions she conveyed the disease to the child. This raises the question of scarlet fever sine exanthema. Let us say that scarlet fever poison can cause a specific angina capable of conveying the disease to the healthy. Certain forms of exanthema of scarlet fever are very evanescent, and in anginal cases may escape observation.

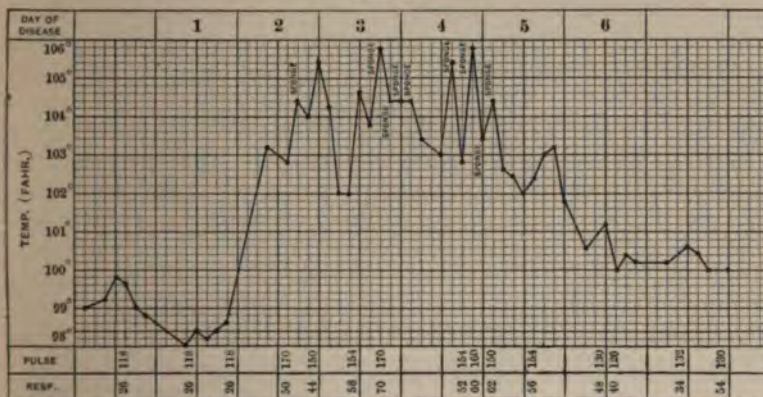
The *period of incubation* has no fixed symptomatology. In many cases the symptoms begin with the appearance of the eruption. The children play about; they have a slight angina, but do not complain. This is apt to be the case with children who are sufferers from chronic catarrh, enlarged tonsils, or adenoids. In other cases the invasion of the disease is a stormy one. There may be an initial convulsion preceded by a sharp rise in temperature. Exami-



nation in such cases may show, previous to the appearance of the eruption, a marked angina or a membranous deposit on the tonsils, but nothing more. Other children suffer from a tonsillitis of moderate severity, a marked febrile movement, and, what is characteristic, attacks of anorexia and vomiting. A chill, followed by fever and vomiting, ushers in a large number of scarlatinal anginas. Occasionally the symptoms of invasion are so mild and evanescent as to escape the notice of even watchful parents. These are the cases in which the first symptom to attract attention belongs to a later period of the disease or to some of the complications. There are thus all degrees in the severity of the symptoms of the period of invasion, varying with the susceptibility of the subject and the virulence of the epidemic.

**General Course of the Disease.**—An attack of scarlet fever takes a certain general course. After the initial symptoms described,

FIG. 28.



Moderately severe scarlet fever; female child four years of age. Normal course. Observed from the outset.

twelve to thirty-six hours elapse, when an eruption or rash appears on the skin: this eruption, though characteristic, varies greatly in intensity, mode of spreading, and distribution. The fever is now very high; the eruption spreads and becomes more intense and general (Fig. 28). At the greatest intensity of the eruption or florescence the fever is highest. In typical cases of scarlet fever the eruption reaches its full development and runs its course within two to six days. At the end of this time it fades, and desquamation begins. The fever subsides gradually, leaving the patient convalescent. The period of invasion is not so sharply defined as in measles, nor is the stage of eruption so distinct and uniform as in that disease. The length of the period of desquamation in both measles and scarlet fever varies.

The malignant cases may at first appear mild. The children are taken with vomiting and a moderately high fever, and the eruption appears. While the eruption is spreading, however, the patients become stupid, and within a few hours after the appearance of the exanthema pass into a state of coma. The urine is diminished in quantity or suppressed, and contains blood, albumin, and casts. The temperature remains elevated (Fig. 29). The pulse is rapid and at times thready. These patients remain comatose and die within a few days (three or four) of the onset of the symptoms. In other malignant cases the affection of the throat and adjacent lymph-nodes is a leading factor in the septic phenomena, while the kidneys show very little participation in the general toxæmia. Such patients will show necrotic pseudomembranous inflammation in the fauces after the eruption is fully developed. The glands of the neck are

FIG. 29.



Malignant scarlet fever; uræmic symptoms from outset. Boy, six years. Sopor increasing to coma; bloody urine. Involuntary passage of urine and feces. Death in three days after onset of symptoms.

involved. The temperature ranges from 103° to 105° F. (39.4° to 40.5° C.), with daily remissions. The patients have a sallow, septic appearance, and are stupid and irritable. The exanthema fades slightly after having been in efflorescence. The lymph-nodes in the neck enlarge to great size. These patients may die in the second week from general toxæmia. Between the normal course and these malignant forms there are all degrees of severity and mildness in this affection.

We shall now consider the various phenomena of the disease.

**The Angina.**—The angina of scarlet fever is limited to the pillars of the fauces, the uvula, the tonsils, and retropharynx. With this there may be a slight suffusion of the eyes. The angina may be simply a slight redness of the fauces and very slight swelling of both tonsils. The lymph-nodes at the angle of the jaw may be



other cases the skin may present minute pustules. There is pruritus in the cases in which the dermatitis is severe. The rash of scarlet fever attains its full development at the end of two or three days. It is then said to be in efflorescence. It remains out a variable length of time, in some cases six days. In other cases the eruption may develop fully in two days and then fade. Cases in which the rash is visible for only twenty-four hours are not uncommon. The appearance of a fading scarlet fever rash is very characteristic if it has involved the whole surface. The skin is dotted here and there by raised papillæ, and appears as if irregularly and lightly daubed with rouge. Even a fading rash may be easily diagnosed by an experienced observer. In mild cases the rash may disappear within twelve hours, leaving no vestige of its presence. In other cases the rash appears only on the lower part of the abdomen and upper part of the thighs.

The eruption on the lower part of the extensor surface of the forearms, and also on that of the legs, is apt to assume a blotchy, roseola-like appearance. Such cases have been mistaken for measles.

Abscesses or furuncles, multiple or single, may involve the skin. In rare cases gangrenous processes have been recorded. A secondary infection may be assumed in all of these cases.

**The Fever.**—In the first few hours there is a rapid rise of the temperature to  $104^{\circ}$  or  $105.8^{\circ}$  F. ( $40^{\circ}$  or  $41^{\circ}$  C.) It remains high with morning remissions until the eruption on the surface reaches its full development. With the fading of the eruption the temperature falls, and within six days, if the case is uncomplicated and typical, becomes subnormal. The patient may show a subnormal temperature for a few days, after which it may rise to the normal. In some cases the temperature may rise very rapidly, reaching its highest point within a few hours. It may then fall to the normal rapidly, though the eruption be still present. Wunderlich and Henoch record cases of profuse exanthema with a mild febrile course or practically afebrile curve,  $101.1^{\circ}$  F. ( $38.4^{\circ}$  C.), falling rapidly to  $100.4^{\circ}$  F. ( $38^{\circ}$  C.) within twenty-four hours. In those cases in which there are complications either in the throat, ear, joints (rheumatism), or serous cavities, the temperature-curve will be influenced accordingly. In other cases, evening remissions may occur instead of morning ones. After the fading of the eruption the fever may continue for days,  $100.4^{\circ}$  to  $102.2^{\circ}$  F. ( $38^{\circ}$  to  $39^{\circ}$  C.), in the absence of any complication. After days or weeks of absence of temperature there may occur a distinct rise and a species of relapse similar to that seen in typhoid fever. This is probably due to a form of secondary streptococcus infection. During the height of the eruption the temperature may reach  $107^{\circ}$  F. ( $41.6^{\circ}$  C.), although in mild cases it may not be over  $103^{\circ}$  F. ( $39.4^{\circ}$  C.). In cases of septic infection, especially of the lymph-

removed; the mucous membrane has a dry, red, beefy appearance. The lips are fissured and bleed easily.

The tongue in most cases of scarlet fever is furred at the outset, and may present a slightly reddened appearance at the borders and tip. Only occasionally do we find the so-called characteristic strawberry tongue. This shows an undue prominence and erection of the papillæ of the tongue, especially at the tip. The tip is red, and with the prominent papillæ gives the appearance of a strawberry or of the tongue of the lower animals (cat). In many cases the tongue later becomes denuded of epithelium and shows the erected papillæ on the dorsum; in others it becomes dry and fissured. The latter condition is seen in the fatal toxic cases.

**The Exanthema.**—The exanthema of scarlet fever, though very characteristic in appearance, varies more than in any of the other exanthemata in mode of appearance, distribution, spreading, and in duration. In the mild cases the eruption is sometimes so evanescent as to escape notice. In other cases it appears only on certain parts of the surface. It may be very discrete in form and punctate. Usually it first appears on the upper part of the chest about the clavicles, spreads down the chest, and around upon the back. At this time it is also seen on the neck, beneath the jaw, behind the ears, and on the temples. It consists of a very finely punctate rose-colored rash. The punctate appearance is the distinguishing feature of the eruption. At the outset this punctate character is best observed on the chest, abdomen, and the nates. If the eruption has in places become confluent, the skin shows a uniform redness. In such cases the punctate character of the rash can best be discovered by studying the skin from a distance in bright daylight. It will then be made out distinctly in those places in which the rash is most recent. A favorite method is to completely undress the patient and study the lower abdomen, the thighs, and nates. In the early cases the punctate character of the rash is apparent on the neck and behind the ears. The appearance of the face at the outset of the disease is characteristic. There is a pallor about the mouth and *alæ nasi*, while the cheeks are flushed with a flame-like erythema. The cheeks do not show the characteristic punctate rash, although flushed either from the fever or intense dermatitis, which involves the whole surface. The eruption spreads from above downward, involving the arms and forearms, hands, and lower extremities. It retains the punctate character wherever it spreads, but loses this characteristic after it has been out for a short time and become confluent. When confluent the rash causes the skin to appear uniformly red and swollen. The skin is roughened in patches by the erection of the papillæ. In other cases, and especially in those occurring in summer, the skin is studded with myriads of minute vesicles. In



other cases the skin may present minute pustules. There is pruritus in the cases in which the dermatitis is severe. The rash of scarlet fever attains its full development at the end of two or three days. It is then said to be in efflorescence. It remains out a variable length of time, in some cases six days. In other cases the eruption may develop fully in two days and then fade. Cases in which the rash is visible for only twenty-four hours are not uncommon. The appearance of a fading scarlet fever rash is very characteristic if it has involved the whole surface. The skin is dotted here and there by raised papillæ, and appears as if irregularly and lightly daubed with rouge. Even a fading rash may be easily diagnosed by an experienced observer. In mild cases the rash may disappear within twelve hours, leaving no vestige of its presence. In other cases the rash appears only on the lower part of the abdomen and upper part of the thighs.

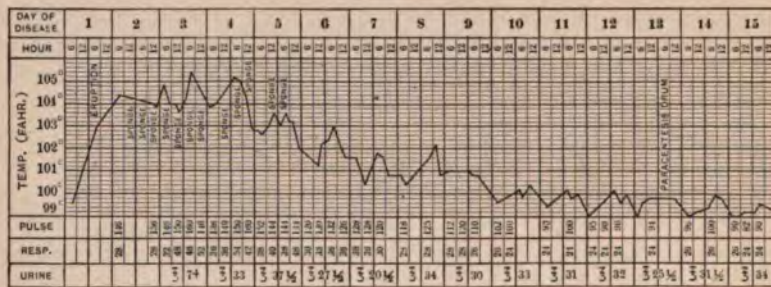
The eruption on the lower part of the extensor surface of the forearms, and also on that of the legs, is apt to assume a blotchy, roseola-like appearance. Such cases have been mistaken for measles.

Abscesses or furuncles, multiple or single, may involve the skin. In rare cases gangrenous processes have been recorded. A secondary infection may be assumed in all of these cases.

**The Fever.**—In the first few hours there is a rapid rise of the temperature to  $104^{\circ}$  or  $105.8^{\circ}$  F. ( $40^{\circ}$  or  $41^{\circ}$  C.) It remains high with morning remissions until the eruption on the surface reaches its full development. With the fading of the eruption the temperature falls, and within six days, if the case is uncomplicated and typical, becomes subnormal. The patient may show a subnormal temperature for a few days, after which it may rise to the normal. In some cases the temperature may rise very rapidly, reaching its highest point within a few hours. It may then fall to the normal rapidly, though the eruption be still present. Wunderlich and Hensch record cases of profuse exanthema with a mild febrile course or practically afebrile curve,  $101.1^{\circ}$  F. ( $38.4^{\circ}$  C.), falling rapidly to  $100.4^{\circ}$  F. ( $38^{\circ}$  C.) within twenty-four hours. In those cases in which there are complications either in the throat, ear, joints (rheumatism), or serous cavities, the temperature-curve will be influenced accordingly. In other cases, evening remissions may occur instead of morning ones. After the fading of the eruption the fever may continue for days,  $100.4^{\circ}$  to  $102.2^{\circ}$  F. ( $38^{\circ}$  to  $39^{\circ}$  C.), in the absence of any complication. After days or weeks of absence of temperature there may occur a distinct rise and a species of relapse similar to that seen in typhoid fever. This is probably due to a form of secondary streptococcus infection. During the height of the eruption the temperature may reach  $107^{\circ}$  F. ( $41.6^{\circ}$  C.), although in mild cases it may not be over  $103^{\circ}$  F. ( $39.4^{\circ}$  C.). In cases of septic infection, especially of the lymph-

nodes, or in streptococcus diphtheria, with infection of the lymph-nodes, the temperature-curve will be of a remittent character, falling and rising once or twice in twenty-four hours, and may retain this character throughout the affection. Uræmia or any affection of the pleura, lungs, or heart will be ushered in by a rise of temperature even if it has returned to the normal. If a complication occurs early in the disease, the temperature will fail to drop to normal with the fading of the eruption (Fig. 30). In cases of otitis persisting through the stage of desquamation there will sometimes be an evening rise, although the ears are discharging freely. In such cases the bone may be involved (mastoid disease). In severe, malignant forms in which symptoms of profound sepsis, such as coma or stupor, are present from the outset, the temperature remains persistently high ( $105.6^{\circ}$  F.,  $40.8^{\circ}$  C.), remitting a degree toward morning. The temperature remains high until the fatal issue (see Fig. 29).

FIG. 30.



Scarlet fever, moderate severity, in a boy six years of age. Shows the delay in the drop of the temperature due to complicating otitis of the right ear at the outset of the period of desquamation.

**Desquamation.**—The period of desquamation begins as soon as the exanthema commences to fade. Generally speaking, since the exanthema first appears on the upper part of the chest and neck we should expect desquamation to begin there. It may be in fine, branny scales, such as are seen in measles; or else, as is most common, the skin peels in larger particles. The hands and feet show the largest scales, and complete casts of the hands and feet are sometimes shed. I have seen the nails shed completely twelve weeks after the attack. The desquamation may be scarcely perceptible. In some cases only certain parts of the extremities, such as the toes or inner portion of the thighs, show desquamation. It is, however, always present. Desquamation in itself is not a pathognomonic symptom of scarlet fever. It occurs in forms of dermatitis which bear no relationship to the disease. It is still a subject of debate whether cases of angina without an exanthema may desquamate. Henoeh is inclined to think this possible. We should remember



that an evanescent, slightly marked exanthema may escape the notice of even the most careful observer.

The duration of desquamation is variable. I have seen the skin desquamate a second time. The severity of desquamation has no relation to the intensity of the exanthema. Some very marked cases of scarlatina desquamate less than those in which the eruption has been faintly marked.

**The Nose.**—The close relationship of the nasal passages to the pharynx facilitates the invasion of bacteria from the throat. The nasal passages become affected simultaneously with the severe angina. There is a severe catarrhal or pseudomembranous inflammation of the mucous membrane. In the so-called septic cases there may be an ichorous discharge from the nostrils. There will be in such cases erosions, and sometimes fetor, with the discharge of necrotic tissue through the nasal passages. Necrosis of the cartilaginous and bony structures may result. In other forms there is a pseudomembranous deposit around the opening of the nostrils extending up into the nasal passages. Casts of the nasal passages may be expelled. The membrane may leave a bleeding surface.

**Ear.**—Duel found the ears affected in 20 per cent. of the cases of scarlet fever. Generally both ears are diseased. Deafness

FIG. 31.



Female child, two and a half years of age. A mild form of scarlet fever complicated in the second week by an otitis.

is frequently a result of otitis. Ten per cent. of those who suffer from deaf-mutism can trace their affliction to scarlet fever. Usually the ears become affected in the third week, although they may be involved at the outset of desquamation. The affection of the ears is ushered in by a rise of temperature and manifestations of pain (Fig. 31). Occasionally tinnitus and deafness are initial symptoms. There may be convulsions or even cerebral symptoms. The onset of ear trouble may be insidious, and not suspected until the purulent discharge makes its appearance. If there are premonitory symptoms, they may precede the perforation by one to three days. Ear

complications in scarlet fever are always of serious moment. Meningitis, sinus thrombosis, and abscess of the brain are among the more serious results, and may result long after the fever has run its course. The onset of otitis usually occurs during the period of desquamation. The patient may be up and about. There is still some redness of the throat, with swelling of the lymph-nodes. There is a sudden rise of temperature to 103° or 104° F. (39.4° or 40° C.). The child begins to vomit food and has headache. At night the child starts from sleep and cries as if in pain. Children do not always locate the pain in the ear. The reason is that the pain occurs before the child is quite awake. The sleep is restless. The muscles of the face and hands twitch in sleep. These symptoms may at times abate. The temperature may fall to the normal and then rise sharply. Any of these symptoms should direct attention to the ear.

The *mastoid* may become the seat of inflammation in the fifth or sixth week. The ears may have been discharging very freely. The child is not, however, free from fever. At times during the day the patient complains of frontal headache, is drowsy, and the temperature shows a rise to 102° or 103° F. (38.5° or 39.9° C.). There is tenderness behind the ear or in front of the auditory meatus. There may be a slight blush above and behind the pinna. In these cases the mastoid may be the seat of suppuration. There are forms of otitis which occur on the eighth day of the disease. The temperature does not fall to the normal. The patient has begun to desquamate, but the temperature remains elevated a degree or more and takes fully three or four days longer to fall to 99° F. (37.2° C.) in the rectum than in an uncomplicated case. At the eleventh day of the disease pain is complained of. The drumhead is found to be bulging. An insidious serous otitis media is in progress.

**The Eye.**—Conjunctivitis may appear in some cases of scarlet fever as a result of a mixed infection. The lachrymal duct is the canal through which such infection travels. Conjunctivitis in cases of gangrenous pharyngitis and rhinitis may lead to panophthalmitis and destruction of the bulb.

**Lymph-nodes.**—The lymph-nodes in various parts of the body enlarge in scarlet fever. Those situated at the back of the neck behind the posterior border of the sternomastoid muscle may enlarge some days before the appearance of the exanthema. At the time of the eruption we may find the lymph-nodes in the axilla, inguinal region, and those at the angle of the jaw, enlarged. In other cases the lymph-nodes, except those at the angle of the jaw, may not be perceptibly enlarged. In some cases the lymph-nodes at the angle of the jaw may enlarge at the end of the second week, with a distinct rise of temperature to 104° F. (40° C.) or more, as a result of reinfection through the tonsils and pharynx. The connective tissue of the neck beneath the body of the jaw is involved in the inflamma-

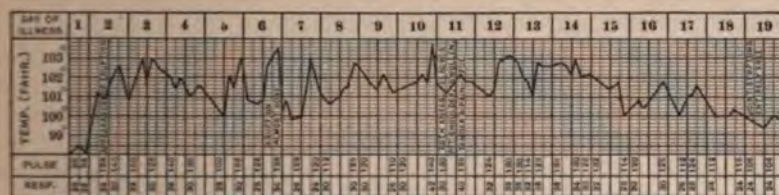


tion of the nodes. In such cases the swelling has an appearance similar to that seen in angina Ludovici. In severe mixed infection the tissues of the neck may become gangrenous. As a result of such severe gangrenous inflammation, phlebitis erosion into the veins and arteries with fatal hemorrhage may result. Retropharyngeal abscess or retropharyngeal adenitis is a sequence of infection of the lymph-nodes. The retropharyngeal abscess in such cases is not as benign as that occurring independently of scarlet fever. In the latter the abscess is apt to involve a chain of retropharyngeal nodes. Multiple burrowing abscesses result. The nodes of the mediastinum may be affected, causing empyema or pericarditis. The mediastinal abscess may cause death by pressure on the trachea, or, by eroding the trachea, burst into it and cause death through suffocation.

**The Mouth.**—Stomatitis always occurs in severe scarlet fever. It may be simply a mild catarrhal process. If there is a pseudomembranous formation on the tonsils, this pseudomembrane may spread to the mucous membrane of the soft palate, and the buccal mucous membrane may also become affected. The tongue is dry and fissured; the lips are dry, fissured, and bleed easily. There may be a discharge of necrotic tissue from the mouth. The soft palate, tonsils, and pharynx may be fused into a necrotic mass, emitting an offensive odor.

**Joints.**—The joints become inflamed in from 2 to 6 per cent. of the cases of scarlet fever. This affection of the joints has been called

FIG. 32.



Boy five years of age, observed from the outset of the disease. Scarlet fever with joint-complications. No cardiac involvement. Recovery.

scarlatinal rheumatism. The joint-affection may, in exceptional cases, precede the exanthema. It appears, as a rule, in the second or third week of the disease (Fig. 32), and is therefore one of the manifestations seen during desquamation. There may be pain in several articulations. In other cases swelling may occur, with effusion of serum into the joints. These cases retrograde. There may be a complicating endocarditis. In other cases there is suppuration of the joint. An arthritis with streptococci in the joint-effusion results. The streptococci invade the joint through the epiphyses of the bone, and produce a streptococcus osteomyelitis with suppuration of the adjacent joints (Lannelongue, Achard, Koplik, Van Arsdale).

As a rule, suppuration occurs in only one joint. Cases in which several joints are affected are generally septic, streptococci having gained access to the general circulation through a necrotic focus in the throat or pharynx. Such cases are fatal. There are metastases in the lungs, kidneys, pleura, and pericardium, with hemorrhages in the skin and enlargement of the spleen. Periarticular abscesses rarely occur (Henoch). The prognosis is serious in all suppurative cases.

**The Kidneys.**—In scarlet fever, as in most infectious diseases, there may be a mild form of nephritis in the earlier stages. There are a small amount of albumin and a few hyaline casts in the urine. This nephritis is of little significance, and has nothing in common with the severer form which occurs later in the disease. The severe form of nephritis begins as a rule in the third week. It has been known to appear in the sixth week. The frequency of this complication varies in different epidemics. In some, only a small number of cases are affected (5 per cent.). In other epidemics fully 70 per cent. of the cases are thus complicated. Its occurrence cannot always be predicted from the severity of the disease. The mildest cases may develop severe nephritis. The diphtheritic forms of angina are more likely to be complicated with or followed by nephritis. On the other hand, the severest forms of scarlet fever may run their course without marked nephritis. Sørensen has shown that at autopsy the most marked changes may be found in the kidneys, although no clinical signs of the affection have been manifested during life. In 50 per cent. of the autopsies upon scarlet fever patients Friedländer found changes in the kidneys. It was formerly thought that exposure played an etiological rôle in this affection, but this view has been abandoned. Nephritis may develop in cases which have been very carefully guarded from exposure from the outset. Although the symptoms will be detailed elsewhere, it may be here stated that the first symptom is a slight œdema about the eyes and face which spreads to the rest of the body, involving the trunk and extremities, the hands and dorsum of the feet, and the scrotum. In some cases the œdema is not marked, in others the anasarca is extreme. The serous cavities may become the seat of effusion, and there may be hydrothorax, hydropericardium, or ascites. The urine also shows changes very early. The quantity diminishes very rapidly, or it may be completely suppressed. The urine shows the presence of albumin, rarely more than 0.5 per cent. It may be highly colored or smoky, or may be distinctly red in color, owing to the large amount of blood and blood-pigment contained. The urine in cases of partial or complete suppression generally contains a large amount of albumin, blood, hyaline, epithelium, and blood-casts, renal epithelium, and leucocytes. The specific gravity may at first be high, 1.030; later, when diuresis is inaugurated, it may fall to 1.006. All cases do not run their course with anasarca.



Henoch has seen cases without this symptom. The invasion of the affection is sometimes marked either by a rise of temperature or convulsions. The prognosis is good in spite of the very alarming symptoms, such as convulsions and coma, which are seen in some cases. This nephritis usually runs its course in from four to six weeks, leaving the kidneys intact. Sometimes the nephritis apparently subsides, but albuminuria of a very mild or intermittent form persists for months. In fact, many of the so-called cases of paroxysmal albuminuria are probably due to unnoticed scarlatinal nephritis. Finally, the author has seen cases in which the anasarca recurred at long intervals as a result of chronic diffuse nephritis.

**Uræmia.**—Uræmia commonly sets in with a diminution in the whole quantity of urine passed daily. It may supervene without any distinct change in the quantity or quality of the urinary excretion (Henoch). In these cases the changes in the urine follow the appearance of the uræmic symptoms. Uræmia may also appear notwithstanding the passage of an increased amount of urine. The latter mode of onset in uræmia is very uncommon. The early symptoms are vomiting, headache, and slight twitching of the facial muscles. These may subside with the abatement of the nephritis. We may have, however, eclampsia as the first symptom; with tonic or clonic convulsions, unconsciousness, and coma with temporary absence of the reflexes. The respirations are increased, and in most cases the temperature rises. The pulse is small and the skin dry. The convulsions may subside, but the coma may continue. The eclamptic seizures may be repeated. The uræmia may subside, and after a very protracted interval reappear with a repetition of the above phenomena. Mania, melancholia, and aphasia may ensue.

Amaurosis without changes in the retina is a more common condition. The retinitis of Bright's disease is absent in scarlet fever. Litten found a swollen condition of the papilla. Amaurosis may persist in the intervals between the convulsions.

The heart action immediately preceding the convulsions is slow. The pulse may be as low as 40 per minute. During the convulsions the heart action is increased. The respirations may be 60 and the pulse 200 (Jürgensen).

The temperature may be 100.4°–103° F. (38°–39.5° C.), rarely 107.6° F. (42° C.), with an initial chill (Jürgensen).

Uræmia may set in at any time while the kidney is affected.

**The Heart.**—Myocarditis of an acute infectious character is likely to supervene in septic cases of scarlet fever. The changes in the myocardium may also be secondary to changes in the pericardium and endocardium.

Endocarditis of the cardiac walls is more frequent than that of the valves. For this reason murmurs should be carefully observed.

No conclusions as to their valvular origin can be reached until long after convalescence. Endocarditis is uncommon, but is more frequent in this disease than in diphtheria or typhoid fever.

Pericarditis is rare. Muscle murmur is often mistaken for it. If present, pericarditis is usually of the dry fibrinous or serofibrinous variety. It is rarely purulent, except in cases of marked purulent involvement of other organs and cavities, notably the pleura.

Dilatation of an acute character may supervene early in severe cases. In such cases we may have tachycardia or bradycardia. There may be cyanosis. Sudden death is very rare in scarlet fever.

Friedländer has shown that in scarlet fever with marked nephritis and uræmia, the consequent increased arterial tension results in dilatation of the left ventricle, with slight hypertrophy. The weight of the heart is increased 40 per cent. The pulse may be slow and irregular. As the nephritis subsides the tension diminishes and the frequency of the pulse increases. Hypertrophy being the result of long-continued increased tension, can be demonstrated only in extreme cases. Dilatation is rarely so great as to cause death.

**Lungs.**—The lungs may be affected by pneumonia, which is generally of the bronchopneumonic type. Lobar pneumonia as a complication of scarlet fever is rare. Gangrene of the lung may occur in the severe septic cases.

**Pleura.**—Pleuritis as a complication of scarlet fever usually appears in the middle of the second week. It is commonly of the serous variety, but the author has had many cases in which there was an empyema usually of the streptococcal variety. Fürbringer has shown that in 5 per cent. of the cases of pleurisy there is nephritis.

**The Blood.**—There is a diminution of the hæmoglobin, which is marked in cases in which nephritis is present. During convalescence the hæmoglobin increases. Slight leukocytosis is also present in the course of the disease. There may be purpura and surface hemorrhages.

**Stomach and Intestine.**—Vomiting has been mentioned as an early symptom in scarlet fever. It is sometimes repeated in the course of the disease if a cough due to any laryngeal or pulmonary complication exists. Diarrhœa is sometimes a serious complication. There may be a simple diarrhœa, in which an excessive number of movements may threaten the life of the patient early in the disease; or, on the other hand, the diarrhœa may subside without serious results. The diarrhœa may take on a dysenteric or typhoidal type, with severe hemorrhages from the gut. There are some forms of diphtheria of the pharynx, stomach, and large intestine in the septic types of scarlet fever which have been described by Litten.

**Sequelæ.**—As sequelæ to scarlet fever may be mentioned:

**Anæmia**, which may persist for some time.



**Glandular Swellings.**—The lymph-nodes at the angle of the jaw are apt to remain enlarged long after convalescence. The tonsils may remain large.

**Tuberculosis** may follow scarlet fever. It cannot be said that there is any distinct connection between the two diseases. Scarlet fever may leave the patient more susceptible to infection either of acute miliary or chronic tuberculosis.

**Nervous Diseases.**—Chorea has been noted by Gerhardt to follow scarlet fever, as have also rheumatic joint-affections with endocarditis.

Facial paralysis may occur as the result of prolonged otitis.

Psychoses, such as melancholia and mania, have been noted, similar to those following typhoid fever or pneumonia.

**Otitis** may remain with a permanent discharge and consequent deafness or mutism.

The **diagnosis** of scarlet fever in most cases presents few difficulties; but, on the other hand, there is no disease in which the symptoms are more indefinite at times. This is particularly the case with those patients who present an evanescent or partial exanthema and only slight febrile disturbance. In some cases the diagnosis must always remain in doubt. Under these conditions it is better to err on the safe side, and to take all precautions of isolation. The exanthema if partial or not very well marked is likely to be overlooked. The angina, which is the most constant symptom, may be mild. The temperature presents nothing typical as in typhoid fever.

It is good practice in the presence of a localized exanthema of a punctate character on the thighs or lower abdomen or the upper part of the chest, with angina and a slight febrile movement, to consider the case as one of scarlet fever. In all cases of sore throat it is wise not to omit an inspection of the general surface. Although some authors have described the angina of scarlet fever as typical in color, the author has never found this sign of value. In some cases of scarlatinal angina the throat is intensely red; in other cases it is of a pale-pink hue; in still others the throat is only slightly inflamed.

The **enanthema** is not of any service in making a diagnosis. The eruption on the soft and on the hard palate is not characteristic.

**Albumin** in the urine is thought by some to be diagnostic of scarlet fever. There may be marked and unmistakable symptoms of scarlet fever without albuminuria. A simple lacunar amygdalitis may cause it.

We must differentiate the eruption of scarlet fever from that of measles and r  theln, from drug eruptions, and those due to irritants.

**Measles.**—In some forms of scarlet fever the eruption on the forearms has a blotchy appearance. Near the wrist-joint the author

has seen it closely resemble the eruption of measles. In these cases the punctate character of the eruption elsewhere on the surface, and the presence of angina, will assist us, in the absence of any enanthema on the buccal mucous membrane, in coming to a conclusion. In measles the diffuse localization of the exanthema on the face, the conjunctivitis and bronchitis, will aid us. In scarlet fever parts of the face, such as the alæ nasi and the region of the mouth, are free from eruption, while in measles these localities are affected by the exanthema.

**Rötheln.**—Scarlet fever is most frequently mistaken for rötheln, and *vice versa*.

In *rötheln*, when the eruption is punctate, it is invariably discrete. There is never the severe dermatitis with swelling of the skin found in scarlet fever. In *rötheln* the lymph-nodes are more constantly and generally swollen behind the sterno-mastoid, in the axillæ and groin. The throat is but slightly reddened. *Rötheln* presents a normal temperature or at the most a temperature at the outset of the eruption of 101°–102° F. (38.3°–38.8° C.) or even 103° F. (39.4° C.), which rapidly subsides to the normal, although the exanthema may be spreading.

**Drug Eruptions.**—Following the administration of quinine some children, like some adults, develop an eruption which closely resembles that of scarlet fever. In the presence of an angina and fever it may be difficult to exclude scarlet fever. Antitoxin of diphtheria, antipyrin, and belladonna also cause a rash closely resembling that of scarlet fever. It is well in such cases to discontinue the drug, and after a few days, the eruption having disappeared, to administer it again. If the patient be susceptible, there will be a repetition of skin symptoms. Kerosene rubbed on the surface will cause a punctate eruption the exact counterpart of a scarlet fever eruption. Among the poorer classes, with whom petroleum is popular as a general remedy, we should think of the possibility of its having been used. If that has been the case, the skin will have a distinct odor of kerosene.

**Prognosis.**—The prognosis in scarlet fever depends largely on the character of the epidemic and the prevalent type of the disease. In some epidemics in New York City the mortality has been exceedingly low—2 to 4 per cent. (J. L. Smith), while in others it has been notably high. In England the mortality varies from 13 to 40 per cent.

Personal idiosyncrasy will affect the prognosis. Some children develop malignant septic types of the disease although the prevailing epidemic is mild.

Cases complicated with severe angina of a septic character do badly from the outset.

Nephritis is a complication greatly to be feared. It may result



in uræmia and death, or the acute may be followed by a chronic nephritis which will ultimately prove fatal.

Otitis may cause serious and even fatal complications, such as brain abscess or sinus thrombosis.

Affections of the endocardium or pleura may prove fatal.

The prognosis of the so-called scarlatinal rheumatism is good. The joints, even if synovitis develops, retrograde as a rule to the normal in from two to three weeks. This may result even if high fever persists for some time during the joint-affection. In the presence of joint-complications it is necessary to be on the lookout for endocarditis or pericarditis. The occurrence of the latter takes place, as a rule, in cases in which there are other signs of septic infection, such as pleuritis and even peritonitis. All these are cases of mixed infection. If synovitis is complicated with such a serious inflammation as pericarditis, the latter is very likely to be purulent, and in that case the prognosis is grave.

We should never pronounce the patient out of danger until the fourth week of the disease has passed without serious complications. A very high temperature at the outset is an element of danger, although not necessarily so. Septic cases with high temperature and pulse above 150 in the first week of the disease are always to be regarded with apprehension.

Lotz shows that the mortality is greatest under the age of one year and between the first and second year. The lowest mortality according to statistics occurs between the tenth and the fifteenth year.

**Morbid Anatomy.—Skin.**—The investigations of Preobrachen-sky show that during the interval from the third day to the fourth week certain changes occur in the skin. These consist chiefly in an erythematous inflammation of the papillary layer, with hyperæmia, hemorrhages, and a diapedesis of erythrocytes and leucocytes. There is an œdematous infiltration of the connective tissue of the skin. The cells of the rete Malpighii show vacuolization. There is also an infiltration of the sudoriparous and sebaceous glands with small round cells. The epithelium of these glands desquamates and necroses. At the time of the eruption streptococci are found in the skin, especially in the vesicles of the sudamina.

The changes in the kidneys will be considered in the chapter on Diseases of the Kidney.

**Bacteriology.**—The parasitic nature of scarlet fever is still a matter for study. Streptococci play a leading rôle in the disease. Micro-organisms have been described in the blood (Hallier, Klebs, Tschamer). Others have seen plasmodium-like protozoa in the blood (Pfeiffer, Doeble).

Streptococci have been found in the throat membranes (Löffler), in the joints (Litten, Heubner, Koplik, Van Arsdale), and in various

viscera (Fränkel, Freudenberg). Streptococci have also been found in purulent foci of the joints and pleura (Raskin), and in the kidneys, in cases which have succumbed to fatal nephritis (Babes). In septic forms of scarlet fever these streptococci exist in the circulating blood (Babes, Lenhartz, Feer). Streptococci have also been found in the cerebrospinal fluid and bone-marrow (Baginsky). Bacteriologists, however, are not willing to assign to these streptococci anything but a secondary rôle, because they present no features which distinguish them from ordinary *Streptococcus pyogenes*. Kurth found that some of the streptococci, the so-called conglomerate-forming streptococci, were of a virulent type. Bretonneau, Hensch, and Heubner have always distinguished the diphtheria of scarlet fever from true diphtheria. Sørensen describes the membranous formations of scarlet fever as milky, yellow, smeary deposits which cannot be peeled from the parts. The membrane seems to penetrate into the mucous surfaces. Ulcers form, and the tonsils, soft palate, uvula, and nasopharynx become a necrotic, sloughing mass. Scarlatinal diphtheria is pre-eminently an inflammatory process with high fever, swelling of lymph-nodes, and suppurations in different parts of the body. If the larynx and trachea are affected, the bronchi rarely become involved. The contrary is true of Löffler diphtheria. In the latter the membrane can be peeled from the surface of the mucous membrane. The membrane is rich in fibrin, and spreads more on the surface and not in the depths. True diphtheria is followed by paralyses. A peculiarity of scarlet fever is that it may occur sporadically for years and yet not become epidemic. This is in contrast to what occurs in measles. In the latter disease the affection may disappear almost completely and suddenly reappear in epidemic form (Hensch, Johanessen, Feer). Epidemics of scarlet fever are less common than those of measles.

**Treatment.—Prophylaxis.**—The diagnosis of scarlet fever once made, the patient should be isolated from the rest of the family. If several children are affected in the same family, these children should be separated and not placed in one room. Otherwise reinfection will occur. The clothes worn just prior to the illness should be sterilized in steam and then aired in the sun. Sufferers with angina who have been about the patient should not be allowed to come into contact with the healthy. All the children of the family should be kept from school. During the illness the bedclothes and linen of the patient should be put into a 1 : 5000 solution of mercuric chloride, prior to being boiled and dried and aired in the sun. The sick-room must be kept well ventilated. There is no advantage in keeping the temperature of the sick-chamber too low. The author has found a temperature of 68° F. (20° C.) comfortable for the patient and those about him. Sunshine and fresh air are of more value than a room uncomfortably cool. If possible, it is well



to spray with some simple cleansing solution morning and evening the throats of any children of the family who are not affected.

The physician should take off his coat and vest and put on a linen robe of some kind before entering the sick-room. On his departure he should leave this robe outside the sick-room, or, better still, outside the window of an adjacent room. If the physician wears a beard, he should wash his face in a 1:2000 solution of mercuric chloride after leaving the patient. The hands should also be scrupulously disinfected. When he returns home he should make a complete change of clothing before visiting other patients. Carpets and superfluous furniture should be removed from the sick-room. The hanging of sheets wet with disinfectants in the door of the sick-room is not essential.

Those about the sick should have no intercourse with the healthy, nor should they go through the house. Meals should be carried by others to some neutral spot.

After convalescence the question of the disinfection of the sick-room and its occupation by others arises. It must be confessed that at present we are in possession of no absolutely sure method of disinfecting a room after its occupancy by a scarlet fever patient. We may adopt one of two methods. The cracks and spaces in the windows and doors are closed with strips of paper glued over them. The disinfectants, preferably a large quantity of binoxide of manganese, table salt, and sulphur, are placed in the centre of the room. The sulphur is then ignited and the doors sealed. Formalin is also effective. After twenty-four hours the room is opened and aired, and the floors and walls are scrubbed with 1:2000 corrosive sublimate. In hospitals the scrubbing is sufficient. The floor and walls about the bed occupied by the patient are scrubbed, and also the bed. The mattresses are steamed in a sterilizer constructed for the purpose. In families it is best to destroy or burn all bedding of hair. Rugs may be aired and disinfected by steam at the establishments equipped for the purpose.

How soon may a scarlet fever patient have intercourse with the healthy? We have no exact data on this important point. Some authors advise that after the termination of desquamation the patient be given a bath of 1:10,000 corrosive sublimate, and then allowed to mingle with the healthy. Others (Baginsky) advise prolonged isolation. It is not always practicable, nor indeed desirable, to isolate a patient for too long a period. Family considerations demand a return to the family circle as soon as possible. In these cases the course first mentioned is the most practicable. In cases which have exhibited a malignant septic form of the disease the author would advise prolonged isolation after convalescence, for the safety of the other children. The urine of a scarlatinal case if there are even mild signs of nephritis, such as albumin and casts, is

believed to be infectious. A recent otitic discharge is thought to be capable of conveying the scarlatinal poison.

The treatment of scarlet fever is largely symptomatic. In an ordinary mild case there is little to do but to regulate the diet, and keep the nose and throat freed from excess of secretion. The skin needs little care. During desquamation it is anointed once a day with a 1 per cent. salicylic acid or boric acid ointment. The urine should be examined daily, for even in the mildest cases severe nephritis is apt to intervene. Vigilance should not be relaxed until after the fourth week. The fever in simple cases needs only the mildest measures. We should remember that the tendency of the fever is to mount until the eruption is fully developed. It then naturally remits. Thus a temperature of 105° F. (40.5° C.) in an ordinary uncomplicated case may not last more than a few hours. In ordinary cases sponging with lukewarm water is efficacious. The aim is not so much to reduce the temperature as to support the nervous system and the heart. In private practice it is well not to resort at once to full baths simply because the temperature is above 104° F. (40° C.). The reverse is true with temperatures which are persistently high for days. In such cases the author resorts to full baths. The patient is placed in a bath at 100° F. (37.7° C.), and the water cooled to 85° F. (29.4° C.). With children it is well not to resort to lower temperatures. This is especially true in the asthenic forms of sepsis. The patients fail to react after the bath, and seem weakened by the excessive cold. The patients remain in the bath about five minutes, and are then taken out. In cases in which the temperature mounts above 105° F. (40.5° C.) we may employ the pack at a temperature of 70° F. (21.1° C.), with much benefit if the reaction is good. The trunk pack may be repeated every one or two hours. The baths above described may be given every four hours. While the patients are in the bath reaction may be promoted by mild friction. Patients with scarlet fever, especially young children, do not bear baths below 75° F. (23.8° C.) well. The old theory that kidney complications are caused by cold baths is not proved. On the contrary, in uremia Kussmaul lays much weight on the beneficial effects of cold packs where hot baths produce untoward symptoms (Baruch).

**Antipyretics** are of little value in scarlet fever, and should not be used unless there is some special contraindication against hydrotherapy. Antipyretics of the coal-tar series especially, weaken the heart in the toxæmia which accompanies scarlet fever.

**Heart.**—The heart is supported in septic cases with high temperature, in the same manner as in other diseases of a toxic nature. Alcohol (whiskey) is not given in mild cases. In considering its administration the kidneys should be taken into account. We wait until the temperature remains persistently high. At the



third or fourth day a constant temperature of 105° F. (40.5° C.) which refuses to abate with treatment calls for the employment of alcohol with other remedies. For a child of from two to five years half a drachm to a drachm of alcohol every three hours is a sufficient dose. Alcohol and digitalis are probably our best cardiac remedies. Caffeine and camphor may also be employed. Strychnine does not seem to do so well in cases in which there is an active myocarditis.

**Throat and Nose.**—In inflammations of these passages we simply keep the parts sprayed with an alkaline solution in order to remove excessive secretion. In this way the patient is made comfortable and the inflammation of the fauces kept within bounds. It is not always possible to spray the throats of the little ones. If there is nasal involvement, the passages may be kept clear by syringing with salt solution. Strong antiseptic solutions or solutions of sublimate or peroxide of hydrogen are of little use if not harmful. Antitoxin of diphtheria is employed if true Löffler diphtheria coexists. In the streptococcic or most common form of pseudomembranous inflammation we have no remedy which acts directly on the inflammation. Antistreptococcic serum has not given encouraging results.

**Lymph-nodes.**—The lymph-nodes, especially in the region of the angle of the jaw, are, if swollen, treated with local cold applications. This frequently affords much relief. Unless distinct fluctuation exists, we should avoid incision of the lymph-nodes of the neck. The author has seen these nodes incised at the beginning of the second week in septic cases, with very unsatisfactory results. Pus is not found in such cases, but only foci of necrosis, which are best left to nature until the patient regains strength. Later in the disease such nodes may suppurate and need incision.

**Nephritis.**—The treatment of nephritis is elsewhere described in detail. The lines of procedure are indicated here. Headache, vomiting, and convulsions are treated with hot baths, and by the continuous irrigation of hot saline solution (Kemp) per rectum. The kidneys are apt to be affected from the outset in malignant cases. In these cases the Kemp treatment with saline enemata is most suitable. With young or intractable children the continuous irrigation of Kemp cannot be carried out. In these cases a high rectal enema of normal saline solution (Cantani) is given twice daily or more often if necessary. If general anasarca is present, the patient is given two warm baths daily; or by wrapping him in a blanket which has been moistened with hot water and then wrung dry we may facilitate diaphoresis with hot air. Digitalis in the form of infusion is the most efficient remedy, combined with moderate doses of potassium acetate, tartrate, or citrate. Milk is the exclusive diet. Complete suppression of urine, with blood and all the anatomical

elements of severe inflammation of the kidney, will sometimes be followed by an increased amount of urine. In such cases the treatment just indicated will not be efficacious. The heart must be supported, and watch kept for uræmic symptoms. Opium should be employed with extreme caution—best not at all in convulsions; chloroform inhalations with chloral per rectum are preferable. Saline enemata at 108° F. (42.2° C.), diuretin, and nitroglycerin are applicable in those cases in which there is suppression of urine.

**Otitis** is sometimes first indicated by spontaneous perforation and purulent discharge. In other cases pain with a sharp rise of temperature will indicate inflammation of one or both ears. Paracentesis is best performed early, even if only slight redness of the drum is present. Later in the disease (fifth or sixth week) both ears may continue to discharge profusely, with an evening rise of temperature. In some cases the author has noted slight frontal headache and drowsiness toward evening. There may be only a slight redness over the mastoid of one or both ears. It is best not to temporize in such cases, but to advise opening the mastoid process to insure drainage and avoid sinus thrombosis or cerebral abscess.

Complications in the lung, such as bronchopneumonia, are treated on general lines. We should in all cases be on the lookout for pleuritic effusion. Extensive effusions must be aspirated. In all forms of pleurisy, even if the amount of fluid is not large, but persists, with a rise and fall of temperature, we should introduce a needle to determine the nature of the fluid. Pus should be evacuated from the pleura in the manner directed in the chapter on Empyema.

**Joints.**—Joint-affections are best treated by immobilizing the affected articulations. The patient should be kept quiet, and sodium salicylate in liberal doses administered. If this is ineffectual after a few days, the joints should be wrapped in cotton moistened with oil of wintergreen, and sodium bicarbonate given in very liberal doses (grain x (0.7) for a child of three or four years, four times daily). If synovitis occurs and the fever continues high, the joint should be aspirated under antiseptic precautions, in order to ascertain if pus is present. If this is the case, an incision with drainage is the proper remedy.

## RÖTHELN.

(*German Measles; Rubella; Trousseau's Roseola.*)

Epidemics of this disease have been described by Forney, 1784; Heim, 1812; Hildebrand, 1832; and in recent times by Thomas and Crozer Griffith. It is an acute infectious disease, contagious from person to person through the atmosphere, though not as highly so as measles. It may occur in the same person a number of times,



and may attack those who have had measles. All children exposed do not develop the disease.

**Age.**—The youngest patient in the author's experience was seven weeks old. The affection may occur at any age. The author has met it in adults. It occurs with the same frequency in both sexes.

**Prodromal Period.**—There is a prodromal period, during which there may be a slight suffusion of the eyes, with swelling of the conjunctival fold at the inner canthus of the eye. In two cases observed by the author the lymph-nodes behind the border of the sternomastoid muscle were observed to be enlarged six days before the appearance of the exanthema. There is no fever or constitutional disturbance. The period of incubation is placed by Thomas and Emminghaus at from fifteen to twenty days. Just prior to the eruption there are headache, nausea, and bronchial irritation (Forcheimer, Emminghaus).

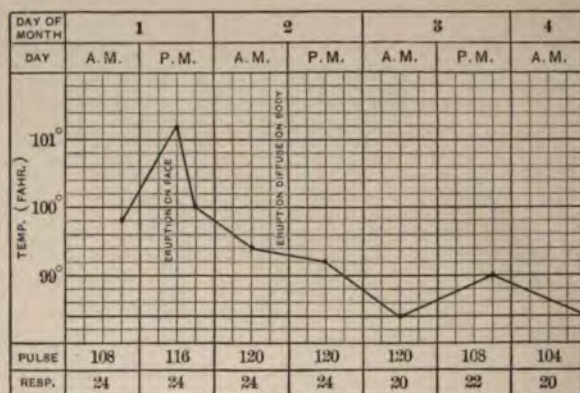
**Exanthema.**—The exanthema resembles that of measles so closely that at the outset it is common for physicians to mistake one for the other. It is also similar in that it is first noticed to appear faintly around the *alæ nasi* and on the upper lips. The exanthema appears first on the face, at the temporal regions, and on the cheeks. It is in some cases preceded by an erythematous blush diffused over the whole face (Emminghaus), which disappears in a few hours, leaving the true exanthema (pre-exanthematic erythema). The exanthema is papular, of a deep rose-red color, and distinctly arranged in crescentic outlines. This arrangement of the papules in circles and half circles can be made out where the eruption is spreading. On the face and neck it gives place to the blotchy appearance characteristic of measles. As a rule, the eruption remains discrete. Edema is rarely present. The papules have been described as of two varieties—one the size of those in measles, and the other punctate (Thomas). The punctate papules have been seen by the author on the upper part of the chest, where the eruption is confluent. They are likely to be mistaken in these cases for the exanthema of scarlet fever. In some cases of Thomas and of the author the punctate papules only were present over the whole trunk. There is an absence of the intense dermatitis seen in scarlet fever, and the individual roseolar spots have the outline above referred to. The exanthema, while fading on the face and chest, spreads slowly on the extremities. The exanthema remains discrete where it is spreading. It remains at its efflorescence on the face and trunk from a few hours to a day, when it begins to fade first from the face, and then from the trunk. A patient may present a perfectly normal skin twenty-four hours after the appearance of the eruption. Evidences of the eruption may remain on the trunk and skin for two or three days. The skin then may present bluish or brownish crescentic spots in place of the original

exanthema, similar to what is seen in simple erythema. Four days after the eruption has appeared the skin in most cases will have a normal hue. There is no pigmentation or discoloration as in measles.

**Desquamation.**—Desquamation is not always apparent. It is possible in exceptional cases to see a very slight desquamation only at the upper part of the thorax or inner portion of the thighs.

**The Eruption on the Mucous Membranes.**—In r  theln the eruption on the mucous membranes does not resemble the exanthema of the skin. There is an eruption in the mouth, but it is not characteristic. There is a mild injection of the conjunctiva, a redness of the fauces, and perhaps a slight cough. Coryza, photophobia, and bronchitis are absent. The mild angina and the injection of the conjunctiva resemble what is seen in la grippe. Thomas and

FIG. 33.



Temperature-curve of a case of r  theln in a boy six years of age. Observed from the outset.

Emminghaus have described an irregular, spotted, streaked appearance, with small grayish miliary vesicles, on the soft and the hard palate. Gerhardt has described a spotted hemorrhagic eruption on the palate, and Forcheimer an irregular macular rose-red eruption on the soft palate. None of these is constant or characteristic of r  theln, but all are found in other affections. The *buccal mucous membrane*, however, is absolutely free from eruption of any kind, and in this fact we have a valuable diagnostic distinction between this disease and measles. In a small percentage of cases a few red stellate spots on the buccal mucous membrane have been seen by the author. In no case, however, was the measles spot with its bluish-white central speck present.

The **temperature** may at the outset be 99.8   F. (37.5   C.) in the rectum, and continue at this point throughout the disease. It may



be 102° F. (38.8° C.), rarely higher. The temperature is highest at the outset when the exanthema appears on the face (Fig. 33). It falls rapidly within a few hours by a sort of crisis. Meanwhile the eruption may spread to the lower extremities.

**Lymph-nodes.**—The author has observed a number of cases with especial reference to the lymph-nodes. Before the appearance of the eruption the nodes behind the sternomastoid and angles of the jaw may be enlarged. At the time of appearance of the exanthema the nodes of the axilla, bicipital groove, and groin become enlarged to the size of a bean or larger. The nodes may remain enlarged for weeks after the eruption has disappeared.

The spleen is not enlarged.

**The Genitals.**—In one case the injection of the vulvar mucous membrane caused temporary dysuria.

**Complications.**—Rötheln is such a mild disease that complications are rare.

**Prognosis.**—The patients recover rapidly.

**Diagnosis.**—The diagnosis of rötheln should not present any difficulties. It is most likely to be confounded with measles, scarlet fever, and erythematous eruptions.

The symptoms are much milder, and there is an absence of the specific buccal enanthema of measles. Measles does not, as a rule, present simultaneous lymph-node enlargements all over the body, such as are seen in rötheln.

Scarlet fever presents a severe dermatitis, which is absent in rötheln. There is a marked angina of a progressive type, with high temperature. The general enlargement of lymph-nodes is not so useful a sign, since in scarlet fever the lymph-nodes of the neck may be enlarged at the angle of the jaw, or those in the axillæ and in the groin may enlarge as the eruption develops. In scarlet fever there is a characteristic desquamation.

Erythematous eruptions of the small papular type may resemble rötheln, but the characteristic crescentic outline of the rötheln roseola is absent.

**Treatment.**—Isolation need not be rigid. Children are kept indoors in summer until the eruption has disappeared and the temperature is normal. In the winter months the patients are kept indoors one week from the onset of the disease. The angina rarely requires treatment.

## MEASLES.

(*Rubeola* ; *Morbilli*.)

Measles is an acute infectious disease distinguished by a characteristic eruption on the mucous membranes and skin. It is highly contagious, and is propagated through the atmosphere. The

specific agent has not been isolated. Most people are susceptible to measles, and suffer from at least one attack. Infants up to the age of five months are not so susceptible as at a later period. Newborn infants have been infected by the mother, and the fœtus has been infected in utero. Only the firstborn is believed by Thomas to be immune for the period mentioned. The disease is very infrequent during the first year of life. Bartels calculates the occurrence at this time at 5 per cent. of the total number of cases. The author has seen measles in infants under five months of age. Measles is most frequent between the age of one and five years (Bartels, Henoch). It is prevalent in all countries of the globe; climate or meteorological conditions seem to have no influence upon its prevalence either endemically or epidemically.

Measles has a well-defined period of incubation, varying from thirteen to fifteen days (Van Panum). In calculating this period we include the time which elapses from exposure to the appearance of the eruption on the body. It will be seen later that this period includes the period of incubation proper, in which absolutely no symptoms, not even fever or malaise, are apparent, and the period of the enanthema on the mucous membrane. The enanthema, which may be accompanied by coryza of mild or severe type, may appear from the ninth to the tenth day after exposure, and lasts from three to five days. Thus while the coryza may be postponed several days or the enanthema may be present for a variable period, the two periods together have a duration of from thirteen to fifteen days. I have seen the enanthema fully five days before the exanthema, and have seen cases of this kind without any manifestations of coryza to signalize the onset of the disease. It is erroneous, therefore, to calculate the period of incubation from the exposure to the onset of coryza, as the latter is variable as to the time of its appearance.

One attack protects the individual from subsequent attacks. Authentic cases of two attacks in the same individual have recently been recorded. By this is not meant a recrudescence of the exanthema after it has once faded. This is also known to occur (Jürgensen). Experiments have proved that measles is highly contagious in the catarrhal stage. Inoculations with the blood (Home) and nasal secretions (Mayr) have given positive results. The period of greatest contagion extends through the period of the exanthema. It diminishes as the exanthema fades, and is thought to disappear gradually during the period of desquamation. Thus though more general in its power to infect, the poison of measles has a shorter period of life than that of scarlet fever. The poison of the latter disease may retain its power of infection months after the disease has run its course. From what has been said, it will be understood that the infection in measles takes place in the vast majority



of cases in the stage of the enanthema (incubation). At this time there may be no coryza.

Infection occurs during the stage of desquamation (Baginsky). If ordinary caution is exercised, it is doubtful whether measles is ever carried by a healthy individual to a third person as scarlet fever is. Baginsky records an epidemic caused in this manner. The poison does not adhere to articles of furniture and wearing apparel with the same tenacity as in scarlet fever.

### The Ordinary Type of the Disease.

The ordinary simple type of measles is that which runs its course without any complications or sequelæ. There is a prodromal period, which includes the period of incubation before the appearance of the enanthema on the mucous membrane of the mouth. During this period it is well established that there are no clinical symptoms whatever—neither fever nor malaise. At the time of the appearance of the enanthema on the mucous membrane the patient begins to feel slightly ill. The symptoms may be only a headache or a slight disturbance of the stomach. The author has noted in some cases a rise of a degree or more in temperature toward evening. There are at this time slight injection of the eyes and general lassitude. Coryza is not pronounced. The patient during the first days of the enanthema, and by this is meant forty-eight to seventy-two hours before the appearance of the exanthema on the skin, presents few signs of illness. If, guided by the very faint redness at the inner canthus of the eyes, we look into the mouth, a few spots of a very characteristic eruption are seen on the buccal mucous membrane. This eruption is pathognomonic of the invasion of measles, and will be later described as the enanthema. After forty-eight to seventy-two hours, and in some cases a longer period, there are coryza, cough, and conjunctivitis. There is a slight febrile movement, varying in intensity in different cases. The exanthema now appears, and is first noticed at the temporal region of the face and the *alæ nasi* as a macular rose-red spotted eruption, which becomes papular later in the course of the disease. The face and scalp are now fully covered by the rose-red irregularly shaped papules, which next appear in rapid succession on the back of the hands, forearms, anterior part of the trunk, back, and lower extremities. This order of the appearance of the exanthema is not always maintained. In some cases, as pointed out by Rehn, and verified by the author, the eruption may first appear on the back. It is, therefore, advisable to examine the patient in a nude state.

The eruptive stage of measles generally lasts three or four days, during which the patient has an exacerbation of all the symptoms of the stage of invasion. There are intense photophobia, active

coryza, and a croupy cough as a result of the invasion of the laryngeal mucous membrane by the enanthema. The bronchi are also affected, and there are symptoms of acute bronchitis. Even very mild cases of measles show laryngeal and bronchial involvement. At this stage the exanthema on the skin is general and profuse, and in places confluent. The patches of healthy skin are crescentic, owing to the peculiar conformation of the papules. In some mild cases the rash may be very diffuse, but in others discrete. In the mildest forms of measles the rash closely resembles in the latter respect that seen in *rötheln*.

The fever reaches its height when the eruption on the skin is fully developed. If the mucous membrane is inspected at the height of the skin eruption, it will be seen that the enanthema becomes diffuse before the eruption of the skin is fully developed. The mucous membrane of the mouth is diffusely inflamed and studded with bluish-white specks which rapidly disappear or desquamate. The eruption on the skin persists for three or four days and then begins to fade. With disappearance of the eruption the general symptoms abate. The fever remits, and the temperature becomes normal by gradual morning remissions. The coryza, cough, and photophobia lessen, and the patient passes into the convalescent period. Desquamation begins when the pinkish hue of the eruption has disappeared. This stage continues until the last vestige of pigmented spots on the skin has disappeared. As a rule, it is completed two weeks after the exanthema has made its appearance. Desquamation is never absent in measles (Crozer Griffith), but it may be difficult to detect its presence. The epithelium is shed in the form of branny scales. Desquamation is best seen on the anterior part of the chest, shoulders, and inner surface of the thighs. In uncomplicated cases it is not attended by constitutional symptoms.

**The Temperature.**—Measles presents no characteristic fever-curve. The invasion is rarely signalized by a chill. There may be a slight sensation of chilliness. The prodromal period before the appearance of the enanthema is not marked by fever. The period of the enanthema presents a slight temperature with morning remissions to normal. When the eruption appears on the skin the fever increases, and reaches its height after thirty-six hours, at the time of the full development of the eruption. The temperature continues high with morning or evening remissions for from one and a half to two and a half days, and then subsides, and disappears in from twenty-four to thirty-six hours after desquamation has set in. The temperature may reach  $104^{\circ}$ – $105.8^{\circ}$  F. ( $40^{\circ}$ – $41^{\circ}$  C.) without complications. During the stage of desquamation the temperature is not elevated unless complication exists in the lung or elsewhere (Fig. 34).

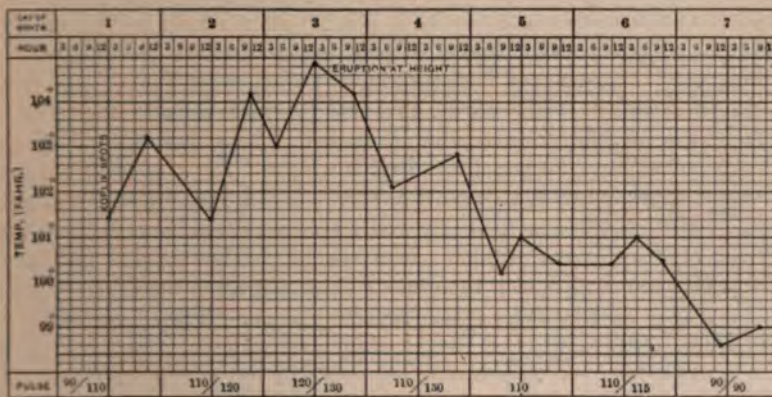
I have sketched the type of disease which is not complicated by serious affection of the viscera and which has no sequelæ. On



account of variations from the simple type just described, measles is one of the most dreaded diseases of infancy and childhood.

In fatal cases occurring during the first two years of life the lung is generally involved (Henoch). The appearance of the eruption is ushered in with a convulsive seizure or a chill. The pneumonia appears as the eruption reaches its height, and within two weeks either proves fatal or else leaves the patient weakened or the subject of an empyema. The infection of the kidneys may be so severe as to prove speedily fatal, or there may be severe mastoid disease. On the other hand, there are cases of measles of a type so mild as to cause little constitutional disturbance. The fever is very mild and evanescent, and present only at the outbreak of the eruption, and even at this stage may be so slight as to escape notice. Jürgensen records measles without fever.

FIG. 34.



Uncomplicated measles in a boy of five years.

**The Enanthema.**—This is the eruption which appears on the mucous membrane of the mouth. It differs from the exanthema in respect to location. The enanthema appears in the mouth from three to five days before the appearance of the exanthema. It is accompanied by redness of the pharynx, and of the anterior and posterior pillars of the fauces. The soft palate is studded with irregularly shaped rose-colored spots or streaks. The spots on the hard palate present small whitish, punctate, miliary vesicles. These spots are also found on the otherwise normally colored mucous membrane of the cheeks and on that opposite the gums of the upper and lower molar teeth. They have been described by Flindt in these localities and on the palpebral conjunctiva. Filatow has described a desquamation of the epithelium of the mucous membrane of the lips and cheeks, in the form of minute whitish shreds (Slawyk). A complete series of studies of the enanthema of measles has been

made, and there can, therefore, be no doubt of its existence. In 1896 I published a study of the enanthema on the *buccal* mucous membrane, and on the inner surface of the lips. In this study I showed that the enanthema on the hard and soft palate so frequently described since the publication of Rehn was not peculiar to measles. The spots of rose-colored papules or streaks with the superimposed miliary vesicles are found in r  theln, scarlet fever, and some cases of simple angina. The eruption on the *buccal mucous membrane* alone, however, preceding the appearance of the exanthema on the skin by a period of from three to five days, is characteristic of the invasion of measles. It is pathognomonic of the disease, and occurs in no other known conditions. It is almost invariably present, observations having shown it to be absent in only a very small percentage of cases.

On looking at the mucous membrane lining the cheeks (*buccal*) in strong sunlight, a very characteristic eruption of irregular stellate or round rose-colored spots is seen. In the centre of each spot there is a bluish-white speck. This appearance of a bluish-white speck on a rose-colored background is pathognomonic of the beginning of measles. The speck is sometimes so minute that strong sunlight is necessary to render it visible. The number of specks at the outset may be less than half a dozen. In a short time they become more numerous, and the rose-colored spots become confluent, so that there are diffusely red patches of buccal mucous membrane studded with bluish-white specks. The specks rarely or never become confluent; their color does not resemble that of sprue, nor are they as coarse as sprue accumulations. They are seen on the inner surface of the lips, and are sometimes well marked on the buccal mucous membrane adjacent to the gums of the upper molar teeth. If the finger is passed over the mucous membrane, they are felt to be raised and firmly adherent. They can be rubbed off by force or picked off with forceps. As the exanthema spreads, the enanthema of the buccal mucous membrane becomes diffuse. When the exanthema is at its height and during efflorescence the eruption on the mucous membrane begins to lose its characteristics. The bluish-white specks are washed away by the buccal secretions and leave the mucous membrane diffusely reddened and raw.

By referring to the temperature-curve, it will be seen that the appearance of the enanthema is accompanied before the outbreak of the skin eruption by fever of a low type.

The **exanthema of measles** is a characteristic eruption of rose-colored or purple-colored papules, varying in diameter from 1 millimetre to 1 centimetre, the average diameter being 2 millimetres. They are irregularly circular, or longer in one diameter than another, or shaped like a half-moon. They arrange themselves crescentically.



## PLATE VII.

Fig. I.



Fig. II.



Fig. III.



Fig. IV.



### The Pathognomonic Sign of Measles (Koplik's Spots).

FIG. 1.—The discrete measles spots on the buccal mucous membrane, showing the isolated rose-red spot, with the minute bluish-white centre, on the normally colored mucous membrane.

FIG. 2.—Shows the increased eruption of spots on the mucous membrane of the cheeks; patches of pale pink interspersed among rose-red areas, the latter showing numerous pale bluish-white spots.

FIG. 3.—The appearance of the buccal mucous membrane when the measles spots coalesce and give a diffuse redness, with myriads of bluish-white specks. The exanthema is at this time fully developed.

FIG. 4.—Aphthous stomatitis sometimes mistaken for measles spots. Mucous membrane normal in color. Minute yellow points are surrounded by a red area. Always discrete.





They are at first discrete, but soon become confluent, so that large areas of skin are covered. Here and there are areas of normally colored skin. The discrete papules have a distinctly crescentic arrangement. This is seen on the thorax and thighs. As a rule, the whole face is covered with the eruption, and the skin swollen. The eruption spreads from the face and head to the back of the neck, throat, upper part of the back, chest, and back of the hands and arms. The lower extremities become affected, as well as the palms of the hands and soles of the feet. As a rule, the eruption on the skin is papular; the papules may show at their summit miliary vesicles. They may become confluent and form patches. Hemorrhages may occur in and around the papules (*Morbilli hæmorrhagica*). In these cases petechiæ occur in the course of the exanthema, and persist into the period of desquamation. They should not be confounded with petechial eruptions or purpura, which may appear after the exanthema has run its course. The exanthema in weakly children may be limited in its distribution and not characteristic. Henoch believes that many cases in which the exanthema does not develop in sequence, take a subsequent course which may be severe. If therefore the exanthema should first appear on the back, instead of the face, and spread thence, complications may be expected. Although complications occur with eruptions which are diffuse and very general, the severity of the eruption is no index as to the severity of the disease.

When the exanthema fades, it leaves the skin studded with dirty brownish-colored spots, which have the arrangement of the original exanthema. These pigmented areas gradually fade, and when desquamation is complete they disappear.

Measles may run its course without the appearance of the exanthema on the face. It may be ill-defined and limited to certain parts of the body. It may develop in full intensity and then suddenly fade within a few hours. This occurs in cases in which severe disturbances of the circulation alter the distribution of blood in the skin. In these cases there may be a complication of the lungs or heart, but the fading of the exanthema is not, as is thought by the laity, primarily the cause of any affection of the internal organs.

**The Nose, Pharynx, and Larynx.**—In very young infants severe inflammation of the mucous membrane of the nose and nasopharynx may lead to difficulties not only in breathing, but also in feeding. In these cases membrane rarely develops. If it does appear, it takes the form of a pseudomembranous rhinitis, generally of a diphtheroid streptococcic nature. Its course then may be subacute. The larynx is sometimes severely affected, so that at the height of the exanthema the patient is troubled with a harassing, croupy cough. In some cases the patient becomes almost aphonic. If there is no

obstruction to the breathing, this symptom, which causes great concern, disappears. The larynx may present a pseudomembranous affection of a streptococcic nature. Gerhardt has shown that ulceration of the posterior laryngeal wall may ensue from traumatism to the larynx as a result of repeated fits of coughing. If these ulcerations cause swelling of the mucous membrane, obstruction to respiration may result. The bronchitis which is always present in such cases may cause obstruction of the finer bronchi. On account of inefficient respiratory effort atelectasis and pneumonia may result, with fatal issue.

**Diphtheria** may complicate measles. It may precede the eruption, or may develop at any time during the attack. In all such cases the patient has been exposed to a double infection. In one case in the author's hospital service the patient had recovered from diphtheria two weeks previous to the attack of measles. Three days after the appearance of the exanthema the conjunctiva became covered with true diphtheritic membrane. The larynx then became involved, and stenosis set in within twenty-four hours after the appearance of the membrane on the conjunctiva. The exanthema in these cases is likely to fade rapidly or become hemorrhagic. Cases of diphtheria complicated with measles are rapidly fatal, since the trachea and bronchi become involved. Fatal pneumonia supervenes. On the other hand, the author has seen a croupy cough with dyspnoea, set in three weeks after convalescence from measles. Diphtheria bacilli were found in the pharynx, and yet recovery took place. In this case no pseudomembrane on the pharynx was visible. It is not always possible to decide in a given case whether there is a simple swelling of the mucous membrane of the larynx or a pseudomembranous process. In cases with severe laryngeal symptoms, if no membrane is visible, a culture of the secretions of the pharynx should be made. The temperature-curve does not aid us. Diphtheria may run its course with a low or a high temperature. The pulse is of little assistance in making a diagnosis. There is nothing in the nature of measles which predisposes toward diphtheritic infection.

During convalescence persistent hoarseness or aphonia is not infrequently seen without other disturbances. The voice gradually returns to the normal.<sup>1</sup>

**Bronchitis; Bronchopneumonia; Atelectasis.**—A very serious complication of measles is bronchitis, which may involve the capillary bronchi, causing atelectasis and bronchopneumonia. In the stage of efflorescence the bronchitis at times becomes severe. There are

<sup>1</sup> Prudden and Northrup, in a paper on diphtheria with fatal pneumonia, record 3 cases of fatal diphtheria complicating measles. The diphtheria and subsequent pneumonia were of the streptococcus variety. The 3 cases formed part of a series of 17 cases of streptococcus diphtheria followed by pneumonia.



found on auscultation fine crepitant râles in addition to the very coarse mucous and sonorous râles. At the end of inspiration a fine crepitation is heard, similar to that present at the beginning of pneumonia. There is also subcrepitation at the close of expiration. In these cases the constitutional symptoms are severe, if large areas of lung are involved. The dyspnoea is extreme. Although cyanosis may be present, no areas of consolidation are detected on physical examination. It is reasonable to infer that in all the cases of severe inflammation of the smaller bronchi, areas of bronchopneumonia exist. Auscultation may reveal areas of lung in which the air enters imperfectly. An attack of coughing will open up the bronchi, when air again enters these areas (atelectasis). In young infants and children this form of bronchitis is a serious complication. As a rule, it leads to bronchopneumonia.

The pneumonia which complicates measles, either in the eruptive stage or in the desquamative period, is anatomically usually of the bronchopneumonic type, although the lobar form may occur. The pneumonia is caused by an invasion of the lung tissue by streptococci from the bronchi. A bronchopneumonia may at first be difficult of detection. As a rule, however, it involves a lobe of the lung in a short time. The lower portions of the lung behind are usually first involved, although the upper lobes or middle lobe may in exceptional cases be first involved. When consolidation takes place, the area of lung involved may be as extensive as in lobar pneumonia. A pneumonic process should be suspected if the temperature in the stage of desquamation does not fall to the normal. There is a distinct rise of temperature which varies in intensity, and remits in the morning to become higher in the evening. The cough becomes troublesome, and there is also dyspnoea. In such cases the temperature alone cannot be relied upon for a diagnosis. A careful physical examination will be of assistance. Under two years of age this form of bronchopneumonia is very fatal. As a rule, pneumonia complicating measles terminates, if not in immediate recovery, in a bronchopneumonia which persists for weeks. The temperature may fall almost to the normal in the morning and in the evening rise a degree or more. In addition to the bronchopneumonia there may be pleurisy, with thickening of the pleura and purulent exudate. In some cases the upper lobe of the lung shows signs of unresolved pneumonia for weeks. Emaciation is progressive. All of these cases are not necessarily tuberculous. A tuberculous process may be engrafted on a non-tuberculous bronchopneumonia at any time by infection with tubercle bacilli. In measles there seems to be a predisposition to invasion of the lung by tubercle bacilli through the catarrhal and inflamed mucous membrane of the bronchi. We can reasonably hope for recovery in many of these cases of simple chronic bronchopneumonia. If tuber-

culous glands, which have been dormant before the invasion of measles exist, they form focal points for the development of tuberculosis of the lungs or meninges. Such cases are fatal. Autopsy will reveal recent lesions alongside of old tuberculous foci.

The frequency of infection with tuberculosis varies in different localities. In some epidemics it occurs in 5 per cent. of the cases; in others, 16 per cent. or more are affected (Bartels, Jürgensen).

**The Heart.**—The endocardium is rarely affected in measles. If endocarditis does occur, it is usually an intercurrent affection in a rheumatic subject. Fig. 35 shows a temperature-curve from a case in which rheumatism preceded an attack of measles, and which in turn was followed by endocarditis. Myocarditis may be found in fatal cases of bronchopneumonia. In bronchopneumonia complicated with pleurisy, pericarditis may also be present (Baginsky).

FIG. 35.



Measles complicated with endocarditis in a boy six years of age.

**The Intestines.**—In some epidemics diarrhoea is a frequent complication. The movements are numerous, and watery in character. When the large intestine is involved the stools contain blood and mucus, and tenesmus is present. The season of the year influences the intensity of the infection. In the warm months the diarrhoea may be of a severe type. In cases recorded by Henoch and Thomas, autopsy showed enlarged Peyer's patches and solitary follicles resembling those seen in typhoid fever. No cases of ulceration have been recorded. Jürgensen is inclined to consider the diarrhoea a result of infection of the intestinal mucous membrane. The enanthema appears in this locality early in the disease.

**The Kidneys.**—In many cases of measles, albumin and a few hyaline and epithelial casts are present in the urine. They are the result of a parenchymatous inflammation of the kidney, due to the poison of the disease. A true nephritis, such as is common in scarlet fever, is rarely seen. Nephritis is apt to occur in the severe



cases complicated with bronchopneumonia. There may then be marked albuminuria, blood, and casts of all kinds in the urine, with suppression. On the other hand, nephritis in the stage of desquamation is rare. There is always in such cases suspicion that an infection coincident with scarlet fever may have been overlooked (Henoch). If diphtheria complicates measles, nephritis is likely to be present.

**The Bones and Joints.**—The author has seen osteomyelitis with suppuration of the joints follow measles. Streptococci were found in the pus. In one case bronchopneumonia was an earlier complication. These cases are rare.

**Lymph-nodes.**—If the inflammation of the throat is severe, the lymph-nodes at the angle of the jaw and underneath the body of the jaw may be enlarged. Rarely, however, is the adenitis as severe as in scarlet fever. The glands or nodes in the axillæ, bicipital groove, over the internal condyle of the elbow-joint, and in the groin may be enlarged to the same extent as in rōtheln, as a result of the processes taking place in the skin. Severe infection of the gut may cause swelling of the mesenteric lymph-nodes, which, if not tuberculous, will retrograde after the disease has run its course.

**The Blood.**—Renaud has recently described a condition of leucocytosis in the stage of incubation. This reaches its maximum just before the appearance of the exanthema, and diminishes as it fades.

**The Nervous System.**—It is rare to see convulsions usher in an attack of measles, even of a severe type. In anomalous forms of the disease complicated with pneumonia there may be cerebral symptoms similar to those seen in the latter affection. There may in some cases be a complicating cerebrospinal meningitis with purulent exudate. If tuberculosis is present, the meninges may be attacked, as in any tuberculous infection. French writers have observed neuritis following measles.

**The Eyes.**—Following severe cases of measles, photophobia, spasm of the orbicularis, inflammation of the lachrymal duct, conjunctivitis, ulcerations of the cornea, and amaurosis may result. Hence, even in mild forms of the disease the eyes should be frequently inspected (Eversbusch).

**The Genitals.**—The author has seen dysuria in cases in which the enanthema affected the mucous membrane of the vulva in girls. Henoch records cases of gangrene (noma) of the genital organs.

**The Mouth.**—Inasmuch as the mucous membrane of the mouth is the seat of an active eruption, stomatitis is likely to be present, especially if through carelessness or traumatism the mucous membrane has become infected with bacteria from without. In such cases aphthæ may result. Children in unhygienic surroundings are likely to develop noma of the cheek if exposed to the infection.

**Pertussis** as a complication of measles is occasionally found. As

in diphtheritic infection, there must have been exposure to the contagion of both pertussis and measles, since etiologically the diseases have nothing in common. The danger in the coincident occurrence of measles and pertussis is that bronchopneumonia is likely to develop, and prove a serious if not fatal complication.

**The Ear.**—The external structures of the ear may be affected by œdema and swelling. The external auditory canal may become the seat of painful swelling and diffuse inflammation. Gangrene of the pinna has been noted (Nottingham, Bourdillot). The most common affection of the ear is otitis media catarrhalis. Of 33 cases of severe complicated measles, Tobieitz found otitis of this variety in 16. The frequency of otitis varies with different epidemics. The otitis makes its appearance in the period between the seventh and the twenty-sixth day following the development of the exanthema. Of 22 fatal cases of measles, otitis was found in 19, only 7 of which presented symptoms during life. The great majority of cases of otitis give no pronounced symptoms and end in resolution. These mild cases are the result of the action through the blood of the measles poison on the ear structures (hæmatogenic). The severe cases follow a mixed infection through the pharynx and Eustachian tube. In the pus of acute or chronic otitis, with or without inflammation of the mastoid, the streptococcus, *Staphylococcus pyogenes*, and pyogenic diplococci have been found. The general course of otitis is not so severe as that of scarlet fever. In some epidemics the severe and fatal cases are more common than in others.

**Sequelæ.**—Any of the complications named above may pursue a chronic course. In this sense only are they sequelæ. Chronic blepharitis, blennorrhœa, keratitis, otitis, catarrhal inflammation or ulceration with stenosis of the larynx, septic retropharyngeal abscess, and chronic bronchopneumonia may persist for weeks or months.

The **prognosis** in measles varies with the virulence of the epidemic, the resistance of the individual, and the age of the patient. It is certain that the idea prevalent among the laity, that measles is a comparatively mild affection, is incorrect. In the cases treated in both dispensary and private practice, and at all periods of infancy and childhood, the mortality is 8 per cent. (Breyer). The mortality is greatest during the first year of life, and may vary in different epidemics from 10 to 40 per cent. The lowest mortality seems to be between the fifth and eighth years—6 per cent. (Baginsky). Hospital statistics are of little value to the general practitioner, as the class of cases treated in these institutions give a high mortality-rate. The mortality in hospitals may be as high as 30 to 35 per cent. (Henoch, Fürbringer).

The **diagnosis** will in most cases present few difficulties if the physician follows a fixed routine in the examination of the patient.



The mode of onset, the coryza, the enanthema of the buccal mucous membrane, and the skin eruption are characteristic. If the physician will examine the inner surface of the cheeks and the buccal mucous membrane in every seemingly slight indisposition of children, he will in certain cases be able to predict an attack of measles far in advance of the appearance of the exanthema. In some cases the enanthema appears on the buccal mucous membrane before coryza is present. The inspection of the buccal mucous membrane thus becomes important as a prophylactic measure. Strong sunlight is essential for thorough inspection. Although the bluish-white spots on the rose-red background may sometimes be seen by artificial light, especially electric light, a diagnosis of measles should never be made at night. Cases of influenza closely resemble measles at the outset. These present the injected conjunctivæ, cough, and rose-colored spots on the soft and the hard palate seen in measles. In la grippe, however, the buccal mucous membrane is pale and presents absolutely no eruption. In one of the early grippe epidemics in New York the children showed an ill-defined roseolar eruption on the surface, but the buccal eruption was never present.

**Rötheln** in some cases resembles mild measles so closely that the author has often questioned whether so-called cases of mild measles without rise of temperature, described by authors, were not cases of rötheln. The difficulty in differentiation is increased if measles is prevalent at the same time. The absence of the buccal eruption is a crucial test. Schmid has also laid stress on this point. In some rare cases of rötheln there may be seen an isolated rose-red spot here and there on the buccal mucous membrane, but the bluish-white speck in the centre of these spots is never seen as in measles.

**Scarlet fever** may at times closely resemble measles, especially in those forms in which the eruption on the face is evanescent. In scarlet fever the buccal mucous membrane has a normal hue. The author has seen scarlet fever complicated with measles. In these cases the scarlet eruption appeared first. Within two or three days there was a general recrudescence of the exanthema, with the appearance all over the body of a roseola (the scarlet rash had faded somewhat), coryza, and the buccal eruption. In other cases the scarlet fever eruption on the back of the hands and forearms assumes the blotchy, papular roseolar form of the exanthema of measles. The author has seen a case of this kind in which an expert entertained the possibility of rötheln or measles. The buccal enanthema was absent. The subsequent course of the case proved the diagnosis of scarlet fever to be correct.

The roseola of typhoid is sometimes so abundant as to mislead the physician into mistaking it for the eruption of measles. Measles complicating typhoid at the end of the second week has come under the author's notice. In this case the buccal eruption was profuse.

Antitoxin and drug eruptions may simulate a measles eruption, but the buccal mucous membrane never presents the enanthema.

The roseola of syphilis frequently resembles that of measles so closely as to cause uncertainty in the diagnosis. Here the conjunctivæ may be injected, and there may be a slight febrile disturbance (Sobel). The buccal mucous membrane is pale, and shows no eruption resembling that seen in measles.

The diagnosis of measles thus resolves itself into a recognition of the disease before and after the appearance of the skin eruption. Before the appearance of the eruption there is very little to guide us. Cough, coryza, and fever may accompany an influenza. In these cases the buccal eruption is of great diagnostic value. After the eruption appears, the question narrows itself to the differentiation of measles from rôtheln or scarlet fever, and the recognition of the various forms of erythema, roseola, drug and antitoxin eruptions.

**Treatment.—Prophylaxis.**—As soon as the physician has made the diagnosis of measles or suspects its presence, the patient should be isolated from the other children of the family. Among the poor it is sometimes impossible to do this. The members of the family not directly concerned in the care of the patient should be denied admittance to the sick-room. It is not necessary to cover the door of the room with cloths or sheets moistened with disinfectants. The physician before entering the room should take off his coat and put on some convenient linen gown or bath-robe, so as to completely cover his person. This robe should hang outside the door of the room, so as to be easily accessible. When not in use, it should be hung in the open air. If the physician wears a beard, he should wash it after leaving the patient, for if the patient coughs in the physician's face, he is likely to carry the infection in his beard to the next child visited. Should the measles be complicated with diphtheria, extra precaution is necessary.

**General Treatment.**—A typical case of measles needs little medicinal treatment. We try to make the patient comfortable. The temperature of the room should be about 68°–70° F. (20°–21.1° C.), if possible. The ventilation should be constant and attained by means of opening doors and windows of rooms communicating with the sick-room. It is not necessary to darken the room very much; in fact, Bartels has shown that light and air are necessary to the comfort and well-being of the patient. The author has found that the ordinary yellow window-shade, if drawn over the windows, sufficiently excludes the actinic rays which are irritating to the eyes.

In a typical case of measles a temperature of 104°–104.5° F. (40° C.) may be ignored. It should be remembered that the fever continues only during the period of the eruption. With the fading of the exanthema the temperature becomes normal. It is only in



cases in which there is a high temperature with delirium that medication is called for. It is not uncommon to see children covered with an eruption and with a temperature of 104° F. (40° C.) playing in bed with toys.

The cough will sometimes need treatment. In such cases I am accustomed to prescribe ℥ iv (0.25) of paregoric combined with ℥ ij (0.12) of syrup of ipecacuanha, every three hours. If the patient is kept awake by the cough, a small dose of Dover's powder (grains j or ij) (0.06 or 0.12) or codeine (grain  $\frac{1}{10}$  to  $\frac{1}{8}$ ) (0.006 to 0.008) at night will be sufficient. If the patient is very restless at night and we do not wish to give opiates, grains v (0.3) of trional will quiet a child of five years. Some young children can be put to sleep by a small dose of phenacetin (grains ij) (0.1). In a mild case, especially if there is pruritus or irritation of the skin, there is no objection to sponging the patient once a day with water at 100° F. (37.7° C.), containing some alcohol or a pinch of sodium bicarbonate.

The food should be light. Milk, broths, and, when the fever has defervesced, chicken, soft-boiled eggs, jelly, toasted bread, crackers, rusk (*Zwieback*), and cereals in attractive form, with cocoa, comprise the diet list. Orange-juice or weak lemonade may be given in moderation. Water-ices may be given, if desired.

As soon as desquamation has set in, I direct the body to be anointed every second day with an ointment of washed benzoinated lard combined with 5 per cent. of boric acid. The patient is allowed to get out of bed as soon as the temperature has fallen to normal, and is permitted to go out of doors three weeks after the outbreak of the eruption in the summer and four weeks in the winter months. Before mingling with other children, the patient should be thoroughly washed with soap. It is not necessary to put an antiseptic in the bath.

**The Treatment of Complications.**—BRONCHITIS; BRONCHOPNEUMONIA.—A severe inflammation of the finer bronchi is likely to cause as much fever, dyspnoea, cough, and restlessness as a primary bronchopneumonia. The temperature then rises and continues elevated—104°, even 105° F. (40°–40.5° C.)—with morning remissions. In these cases the temperature must be reduced. I never hesitate to utilize hydropathic measures. The most convenient mode of applying water is by means of compresses moistened with water at 80° F. (26.5° C.). If the patient reacts well, the compresses may be at 67° F. (19.4° C.); if he becomes cold and cyanosed, at 105° F. (40° C.). These warm compresses are at times very soothing, causing the patient to drop into a quiet sleep. It should be remembered that the object of applying the compresses is not always to reduce temperature rapidly, but rather to stimulate the heart and support the patient. Douching the head with ice-cold water, as recommended by some, is a very questionable practice.

The use of the coal-tar antipyretics should be avoided. In lowering the temperature they act as depressants. In severe cases of bronchopneumonia aconite should not be used to lessen the rapidity of the pulse. Caffeine, camphor, strychnine, and digitalis in proper doses are more satisfactory. If a bronchopneumonia be prolonged into the convalescent stage, we should be on the alert for pleuritic effusion. This is especially likely to occur if the pneumonia lasts longer than two weeks. In these cases the symptoms present are similar to those described under Pleurisy, and the treatment is carried out on the same principles.

The LARYNGEAL SYMPTOMS become harassing when there is much swelling or slight erosions of the laryngeal mucous membrane. In such cases an improvised tent should be erected over the crib or bed and filled with steam vapor saturated with thymol or turpentine. Older children can be persuaded to breathe the vapor generated in an open kettle. If symptoms of stenosis appear, it must at once be determined by culture whether a diphtheritic process, a streptococcic pseudomembranous formation, or a stenosis due to simple catarrhal œdema of the mucous membrane is present.

DIPHTHERIA.—Antitoxin is indicated in diphtheria either of the conjunctiva, pharynx, or larynx. A large dose should be given at the outset, on account of the virulent nature of this affection as a complication of measles. We should not be too ready to intubate on the first appearance of stenotic symptoms. Many of these cases improve. The introduction of a tube into the inflamed larynx in measles is not without danger of causing ulcerations of a troublesome type after the measles has run its course. It is well to follow O'Dwyer's advice in such cases— withhold the tube as long as dangerous dyspnoea is absent. The use of apomorphine, tartar emetic, or turpeth mineral, so popular with continental physicians, to expel membrane or secretion, is of doubtful value.

THE EAR.—Otitis should be suspected if there is restlessness and an intermittent course of temperature without apparent cause. Older children may indicate the seat of pain. In some cases it may be necessary to incise the tympanic membrane. The procedure affords relief from pain, and is without ill effects. Pus or a few drops of serum only may be evacuated.

DIARRHŒA requires the same treatment as a primary enteric catarrh.

The care of the EYES, NOSE, and MOUTH should be conducted on general lines. If the secretion is excessive, the eyes may be bathed once a day with a lukewarm weak saline solution. Unless the secretions are excessive, the nostrils should not be syringed or douched. If clots of mucus or pseudomembranous shreds form in plugs, they may be dislodged once a day by a nasal washing with a



suitable hand syringe. The mouth should not be washed more than once a day. This should be done both for infants who are fed artificially and for older children. On account of the great vulnerability of the mucous membrane in this disease the utmost gentleness should be exercised lest aphthous ulceration be developed.

### VARICELLA.

(*Chickenpox*; (Ger.) *Windpocken*.)

Varicella is an acute infectious disease with a characteristic exanthematic eruption. It is distinct from vaccinia or variola, is an affection of childhood, occurring before the tenth year, rarely later, and is transmitted through the atmosphere. It cannot always be conveyed by inoculation, as is the case with vaccinia or variola. It does not protect from vaccinia or variola. Varicella, vaccinia, and variola have been observed to attack the same patient successively at very short intervals. Few children escape after exposure, and one attack does not confer immunity. Varicella is an endemic disease, and does not occur epidemically.

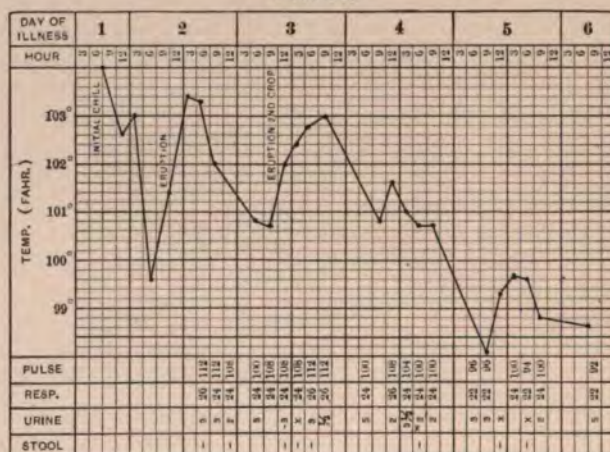
**Incubation.**—Varicella has a period of incubation during which competent observers have noted no disturbances (Hench); others record malaise, coryza, and sore throat. The author is inclined to regard the prodromal period as free from symptoms. The period of incubation is usually fourteen days, but it may be protracted for nineteen days.

The **symptoms** consist of an exanthema, an enanthema, fever, and slight malaise. There may be complications. Previous to the appearance of the exanthema there may be a slight febrile movement and malaise, which in children may pass unnoticed. In cases pursuing a normal course, a chill with a marked rise of temperature may precede the eruption by fully twelve hours. When the eruption appears the temperature gradually falls, unless another crop of papules appears, when there is another sharp rise of temperature. Sore throat and slight malaise may herald the eruption. Previous to the appearance of the rash there may be, as in measles and in varioloid, an erythema of the surface prior to the appearance of the exanthema.

The **exanthema** consists of an eruption of roseolar papules varying in size from that of a pin's head to that of a split pea. They first appear on the forehead and face, and spread to the trunk. In some cases larger blotches appear, but these are of the nature of an erythema, which may precede the eruption of the roseola by a few hours. The roseolar papules have a characteristic violet-rose tint, are raised above the surface, and are sometimes hard to the touch. In a few hours the papule develops on its summit a vesicle, which rapidly fills with lymph. These vesicles become tense, and if the

papule is irregular in shape cover the whole upper surface of the papule. In many places the vesicle at the stage of its efflorescence presents an umbilication which strongly resembles that seen in the vaccinia pock. The contents of the vesicle become cloudy and then yellow; the vesicle is surrounded by a dusky pink areola. In the course of a day or two the cycle is completed, and the vesicopustule begins to desiccate. A reddish-brown scab is developed. Many of the roseolar papules do not develop the vesicle and pustule. While one crop of papules is going through the cycle described above, others appear on various parts of the body. It is characteristic of varicella to have the surface covered with roseolar papules, papules with vesicles, and with pustules, in various stages of development. The papules vesicles, or pustules may be few or very abun-

FIG. 36.



Varicella temperature-curve showing successive rises due to a new eruption of papules and vesicles. Boy aged six years.

dant. In some cases after the scab of the vesicle has fallen off a distinct scar is left, similar to that seen in vaccination, but much smaller; it may persist for years. The skin between the papules and vesicopapules is normal in color.

The soft palate and sometimes the hard palate may show a few isolated papules, vesicles or vesicopustules similar to those seen on the cutaneous surface (enanthema). In most cases there is an angina, an injection of the conjunctivæ or even an enanthema on the ocular conjunctiva (Hench). Thomas records varicella papules and pustules on the nasal and vulvar mucous membrane (Fig. 36).

The temperature is in many cases little raised above the normal. In others it reaches 103° F. (39.4° C.) at the outset of the affection. In rare cases 106.5° F. (41.3° C.) has been observed. As



soon as the eruption is fully developed the temperature rapidly becomes normal. The duration of the fever varies from one to three days. I have seen severe cases in which the high temperature persisted fully a week. The eruption was in these cases accompanied by secondary pustulation.

**Other Symptoms.**—Many infants and children show little constitutional disturbance. In other cases there is lack of appetite with excessive irritability. In others, on account of the profuse eruption in the vulva and around the nates, there is annoying vesical tenesmus and even rectal tenesmus. The latter condition I have seen in a child two and a half years of age, in whom there was a profuse eruption of vesicles in and around the introitus vaginæ, on the nymphæ, and around the anus. There is in some cases a recrudescence of the exanthema in various parts of the body, with rises of temperature.

**Complications.**—Gangrene of the skin with sloughing of large areas has been noted by some observers (*varicella gangrenosa*). The conclusion is inevitable that in many of these cases there must have been a mixed infection.

**Nephritis.**—In many cases there is albumin in the urine to the extent of a trace. Hænoch has described 6 cases of varicella complicated with nephritis on the eighth to the fourteenth day after the appearance of the eruption. In these the eruption was profuse and accompanied by fever; there was œdema with albumin and casts in the urine. One case with fatty liver and moderate hypertrophy and dilatation of the left ventricle resulted fatally. Other authors have confirmed the observations of Hænoch. I have seen slight albuminuria in some cases of varicella.

**Joint-affections.**—I have observed two cases of varicella with swelling, pain, and effusion in one or both knee-joints. In neither was there suppuration. Both cases retrograded, and in a few days the joints became normal. The whole picture simulated what is seen in some cases of scarlet fever. There was no endocarditis.

**Otitis** may occur as a complication of severe cases.

**Pneumonia** is an occasional complication (Fig. 37).

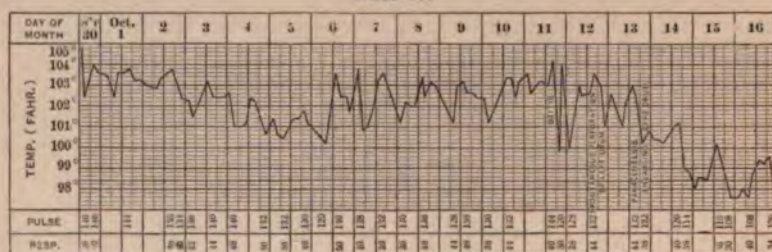
The **diagnosis** of varicella should present few difficulties. I have seen a number of cases in which the eruption was not only very profuse, but the individual varicella vesicles or pustules were also very large. In these cases there may always arise the question of differentiation from the more serious affection, variola or varioloid, especially if an epidemic of smallpox is prevalent. The diagnosis may even in some rare cases remain in doubt (Jürgensen). In varicella the temperature is lower and the rise shorter in duration than in even a mild case of smallpox. In the absence of an epidemic, the mildness of constitutional symptoms,

discreteness of the varicella eruption, and the absence of any œdema of the skin between the vesicles will aid us.

In some cases the eruption of roseola papules on the face and trunk has not the characteristic appearance of vesiculation or pustulation seen in varicella. It is difficult on account of the effects of the scratching of the patient to differentiate the eruption from pustules of a furuncular type. Under such conditions a close inspection of the back may result in the discovery of one or two typical varicella vesicles.

The **prognosis** is very good in varicella, except in neglected cases, in which sepsis may complicate the disease. The very rare

FIG. 37.



Varicella bullosa, pneumónia, otitis media purulenta. Female child aged six years.

cases of nephritis (Henoch) should be borne in mind. In private practice and in a large ambulatory clinic I have rarely seen the severer types of this disease. I agree with Fürbringer in thinking that such cases raise the question of the possibility of an extraneous infection.

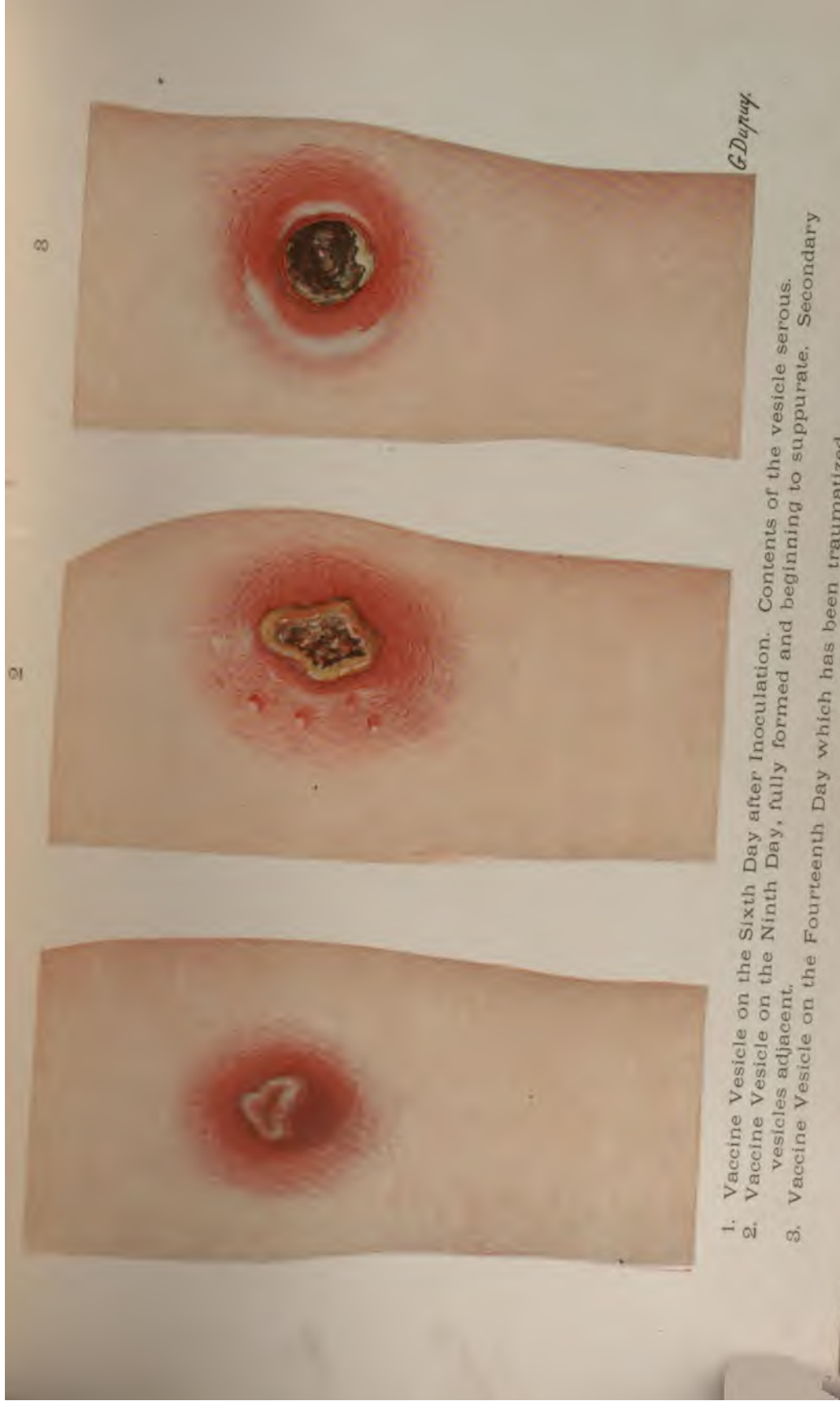
**Treatment.**—Though the course of varicella is mild, the cases should be isolated like those of any other infectious contagious disease. We can never predict the outcome of a number of cases occurring in epidemic form, although individual cases do well. If there are itching and tension, the eruption is covered with 5 per cent. boric acid ointment applied without lint. The children are allowed out of doors as soon as the temperature has become normal, the scabs of the varicella vesicles or pustules have fallen off, and the skin has become normal.

## VACCINATION.

Vaccination is a prophylactic measure against variola practised on the human subject. It gives a certain, though not lasting, immunity against the disease. It is accomplished by inoculating the human subject with the contents of the cowpox vesicle.

Cowpox or vaccinia (vacca, cow) is a specific exanthema which





*G. Dupuy.*

1. Vaccine Vesicle on the Sixth Day after Inoculation. Contents of the vesicle serous.
2. Vaccine Vesicle on the Ninth Day, fully formed and beginning to suppurate. Secondary vesicles adjacent.
3. Vaccine Vesicle on the Fourteenth Day which has been traumatized





occurs on the udder of the milch cow, hence the name. Vaccinia is inoculable from animal to animal, and also on the human subject. It occurs only at the point of inoculation.

Successful vaccination gives the human subject almost certain protection for a long time against vaccinia or cowpox and variola or smallpox.

The essential cause of vaccinia in animals and the human subject has been described by Guarnieri and Kurlow as vaccine corpuscles. These are found in the vaccine vesicle and pustule. They are peculiar, finely punctate, amœbic masses of protoplasm, showing vacuoles. Loudon and Salmon, on the other hand, deny any specific properties to these corpuscles. They think they are simply degenerated leucocytes, and are seen in other simple forms of inflammation.

Edward Jenner (1749-1823) was the first to establish the doctrine of vaccination on scientific experimental data. He was the first to use humanized vaccine—that is to say, to inoculate the human subject with lymph from a cowpox vesicle, and then to utilize the lymph of the vesicle in the human subject to inoculate others. This method has been abandoned. To-day the lymph used is obtained directly from the animal. The lymph is, as a rule, inoculated from animal to animal for several generations. It is just as effective as the lymph of the first animal of the series inoculated. It is called animal lymph or vaccine. The disadvantages of using humanized vaccine are many. First, there is a natural reluctance among some people to vaccinate their children with lymph obtained from the human subject. Apart from the popular belief in the transmission of tuberculosis, scrofula, and other forms of blood disease in this way, it is not always possible to exclude an infection, such as syphilis. The animal lymph can be controlled in its manufacture and produced with all scientific precautions. Animal lymph and human lymph do not differ in the power to confer immunity against variola. The animal lymph should be obtained from the healthy animal in the vesicular stage of the eruption; this is the fourth or fifth day of cowpox. It is preserved by mixing it with three or four times its bulk of glycerin. It may be put up for use on quills or ivory slips in a dry state or in small capillary tubes in the liquid condition. The so-called vaccine pulp, made up of the contents of the vesicle and its epidermal covering, and preserved in glycerin, is not used in this country.

**Age at which to Vaccinate.**—Every infant and child should be vaccinated. There is no contraindication except some acute or chronic illness. Even the hemorrhagic diathesis is no contraindication. Vaccination is best done between the fourth and the sixth month, before teething has begun (Zimmerman). In an emergency, such as the presence of an epidemic of smallpox, the newly born infant may be vaccinated.

**Mode of Vaccination.**—Boys are vaccinated on the left arm; girls,

for esthetic reasons, may be vaccinated on the thigh or calf of the leg instead. The outer surface of the arm, at about the insertion of the deltoid in the humerus, is usually selected. The skin is carefully cleansed with soap and water, washed with alcohol, and dried. With a clean sewing-needle the skin is scarified three or four times in one direction, and at right angles to the first scarifications. We should not cause bleeding, but only expose a raw surface. The scarified area should be about one-eighth of an inch square. The lymph is now rubbed on the scarified area. If quills are used, the vaccine on the quill is moistened with a drop of distilled water before inoculation. Scarifying large areas is likely to cause excessively large pustules, with subsequent severe inflammatory reaction. On the other hand, a small area of scarification may give a very large pustule. In other words, the size of the vaccine pustule does not always depend upon the size of the area of scarification. A mixed infection will give a severe reaction with a very small area of scarification.

**Lymph to Use.**—Either the liquid or the dry lymph may be used. Both are reliable if recently prepared. If the lymph is not fresh, or there is carelessness in its use, the vaccination will be a failure.

**Course of the Vaccination.**—The great majority of vaccinations are very uniform in history. There is an incubation period, during which the wound heals. There are absolutely no symptoms. This period usually lasts three days, sometimes only two, and may be prolonged to four or six days. After this period there is the eruptive stage, ushered in by the formation of flat rose-red papules at the points of scarification. The papules are either oval or irregularly long. On the fifth day a vesicle appears in the centre of the papule and spreads to the periphery. On the sixth day the vesicle takes up the whole papule, has a pearly lustre at the surface, and presents a central umbilication (Jenner's vesicles). The seventh day is the day of efflorescence; the vesicle is filled and tense with lymph, has a rose-red areola and a hyperæmic zone outside this areola; there are itching and tension. On the eighth day the contents of the vesicle become slightly cloudy. On the ninth day the suppuration is pronounced, and on the tenth day the suppuration, swelling, and inflammatory reaction are at their height. At the end of the tenth day there is a retrogression of all the symptoms. The vaccine pustule becomes less angry looking and the inflammatory reaction subsides. A crust forms which may become dry, hard, and fall off, leaving a scar beneath. This takes, as a rule, from ten to fourteen days.

Fever in some cases begins on the fifth day after vaccination. It may be slight and reach its height between the eighth and the tenth day. There may at this time be slight digestive disturbances, such as vomiting or greenish movements.



The areola around the vaccine pustule may spread so as to involve most of the upper part of the arm, or the inflammatory reaction may spread over the entire arm, and sometimes over the back. There may be enlargement of the lymph-nodes in the axillæ. These lymph-nodes may suppurate. If there has been no mixed infection, they retrograde with the pustule.

**Complications.**—Complications may result from traumatism of the pustule, mixed infection (that is, the presence of impurities, such as streptococci or staphylococci in the lymph), lack of cleanliness at the time of maturation of the pustule, and retention of pus in a dressing. The most common complication is an exceedingly severe reaction, with an extensive necrosis of tissue. This may affect the fasciæ or muscular layers, causing large loss of tissue. Among the rarer complications of vaccination is a true septic infection. In these cases there is a history of mismanagement of the pustule, such as traumatism or the compression of the arm by a bandage. Infection which manifests itself in a remittent febrile curve occurs. In one case which came under my notice a few pus-corpuscles appeared in the urine, the elbow-joint and other joints became painful and swollen, and suppuration in the joints resulted. These cases are fatal. There is a true osteomyelitis of the heads of the bones, with formation of pus in the joints. In other cases the child may by scratching inoculate itself elsewhere, either on the arms or even lips and eyelids; the latter condition has come to my notice. It forms a very painful and severe complication. Erysipelas may set in early or late in the history of the vaccination. It may spread down the arm and forearm on the trunk and may endanger the life of the patient. In other cases there may be suppuration of lymph-nodes. In susceptible subjects a rebellious eczema may appear as a direct sequence of the vaccination. Among the complications may be mentioned axillary adenitis, hemorrhage into the pock (trauma), exuberant granulations, and keloid of the scar. Tetanus may result from infection of the wound with the bacilli of tetanus which may be present in uncleanly dressings.

**Generalized Vaccinia.**—This is a general eruption of vaccine pustules, which in rare cases appears from the third to the seventh day over the whole trunk and extremities. It is really a generalized cowpox, similar to the generalized eruption in the exanthemata. D'Espine and Jeandin describe cases in which there can be no doubt of the absence of infection of the surface by the nails or otherwise. The prognosis in these cases is good; there are no severe symptoms, and the fever is slight.

The **management** of a normal case of vaccination is important. We should protect the vesicle from traumatism by means of some simple contrivance, such as a shield. If the areola is angry looking and the redness and swelling severe, we may paint it once a day with

compound tincture of benzoin. This is very soothing and protects the surface from friction. If complications occur, they should be treated on surgical principles. Above all, there should be no retention of pus by any form of dressing. Dressings which seal the vaccine pustule hermetically from the air cause retention, and are therefore dangerous. Sepsis as described above is not the result of vaccination, but of subsequent mismanagement.

**Vaccination Eruptions.**—The eruptions which follow vaccination or occur while the pustule is still in course of development are of interest. Sobel has made an exhaustive study of these eruptions. Fully 14 per cent. of the vaccinations are followed by more or less generalized eruptions. They appear while the local site of the vaccination is open or as late as eight weeks after the primary inoculation, but most often between the ninth and the fourteenth day after inoculation. They have no relation to the size or severity of the local pustule, which may be normal. Among the types of eruptions are the erythematous, urticarial, papular, vesicular, pustular, morbilliform, bullous, pemphigoid, and scarlatiniform. Auto-inoculation by scratching generally occurs an inch or two from the original site, but it may occur elsewhere, as on the eyelid or conjunctiva. The most common type of generalized eruption is undoubtedly the urticarial in its various forms. These include wheals, papules, bullæ, and vesicopapules. The morbilliform are easily differentiated by the absence of fever and coryza and other signs of measles. The scarlatinal forms cause great uneasiness and elevation of temperature. These cases should be observed for urinary complications and subsequent desquamation, in order to exclude scarlet fever. Among the rarer types are the ecthymatous eruptions.

**Revaccination.**—Vaccination should be repeated after the lapse of ten years, and every five years thereafter. During an epidemic, every one who has not been revaccinated should be vaccinated. Immunity to variola diminishes as we reach the termination of the first decade after the first vaccination. If the revaccination runs a typical course identical with that of the original vaccination, immunity is generally lasting.

## OTHER SPECIFIC INFECTIOUS DISEASES.

### TYPHOID FEVER.

(*Abdominal Typhus*; *Ilcotyphus*.)

**Occurrence.**—Of 84 cases of typhoid fever treated by the author, 38 were of the male and 46 of the female sex. The ages were as follows: 1 was of eighteen months, 4 were three years, 9 were four years, 41 were between the fifth and the tenth year, and



the remaining cases ranged up to the fourteenth year. Thus 16 per cent. occurred before the fifth year, and fully 50 per cent. from the fifth to the tenth year.

**Typhoid Fever and Pregnancy.**—According to Etienne, quoted by Morse, the fetus in utero is born prematurely in 70 per cent. of the cases of typhoid fever in the mother. The causes of the abortion are much the same as those which obtain in pregnant women suffering from any infectious disease. The high temperature, the toxins in the circulation of the mother, and the death of the fetus, all contribute to cause miscarriage. Morse believes that the death of the fetus is chiefly instrumental in causing its expulsion. Of 12 abortions, 9 were stillbirths, 2 lived four and 1 five days.

**Fœtal Typhoid.**—Though doubt has recently been cast upon the results of Chantemesse and Vidal, there is good reason for believing that the *Bacillus typhosus* (Eberth) can pass through the healthy or diseased placenta from mother to fetus (Morse). This has been proved by experiments in animals (Vidal, Chantemesse). The typhoid bacillus has been found in the organs of the fetus and in the amniotic fluid.

The anatomical changes found in the fetus affected by typhoid fever are not identical with those seen in the adult. This is due to the fact that the infection of the fetus is hæmatogenous, which explains the high fetal mortality. The spleen is sometimes though not always enlarged. The changes in the gut are not characteristic, being confined to a few enlarged follicles. The liver may be enlarged, and the kidney may show hemorrhages.

**Infantile Typhoid.**—It has recently been contended that typhoid fever is rare in the infant or the child under two years of age. With the improved methods of laboratory diagnosis of typhoid fever we may shortly be in a position to determine the relative frequency of the disease in the newly born and the young infant. Typhoid fever certainly occurs under the age of two years. As Crozer Griffith has pointed out, we should think of the possibility of its presence in every case of continued remittent fever of the nursling not to be explained on other grounds. Of 331 cases, 9 under two years of age were diagnosed by Hensch as typhoid fever. Among others who report cases are Ollivier, Noyes, Northrup, and Bell. I have seen only 2 cases under two years. Blackader, in a recent series of 100 cases, met 4 under two years of age. Gerhardt reports a case in an infant twenty-five days old, and Blumer 1 in an infant five days old. These cases may be regarded as either congenital or post-natal typhoid.

**Morbid Anatomy.**—It has been stated that when the fetus in utero is affected with typhoid fever the process is in the nature of a hæmatogenous infection, and that there are few if any characteristic anatomical changes. In young infants and children

the changes in the gut so characteristic of adult cases are not always seen in their full development. The solitary follicles and Peyer's patches are enlarged, but ulcerations are seen only here and there, and seldom lead to perforation (Monti). On the other hand, in older children the changes in the gut closely resemble those of the adult, as has been shown by Henoch. The mesenteric lymph-nodes, especially those in the vicinity of the ileocaecal valve, are enlarged. The remaining changes resemble those seen in the adult subject.

**Symptoms.**—The invasion of the disease in young children is rarely with a chill. More frequently there are indefinite chilly sensations and mild general malaise. There are headache, pains in the limbs, vertigo, and in many cases vomiting. The symptoms of the period of invasion are so very indefinite in infants and very young children that cases sometimes escape diagnosis.

In other cases, after a few days of malaise the cerebral symptoms become marked. The headache is augmented by delirium at night, especially in older children, and stupor is present. In younger children the period of invasion may simulate a pneumonia. In fact, these cases begin as pneumonia, and it is only on careful consideration of the clinical symptoms—the predominance in a few cases of cerebral symptoms or the enlarged spleen, and the presence of roseola later on, with the elevation of temperature—that we are led to think of typhoid fever. In some of these pneumonic cases there are none of the characteristic features of typhoid. There is no roseola, no splenic enlargement, no epistaxis, but there may be diarrhoea. During an epidemic only the systematic examination of the blood (Widal) will reveal these cases. Such a case is the following: A child, five years of age, was admitted to my hospital service with an indefinite previous history. Temperature  $104.6^{\circ}$  F. ( $40.3^{\circ}$  C.), pulse 140, and respirations 30. There was apathy, also a broncho-pneumonia in the upper lobe of the left lung. This case gave a very positive Widal reaction early in the disease. The spleen became palpable four days after admission. In another case, of a child four years of age, signs of a lobar pneumonia of the upper lobe of the left lung were present without any roseola, enlarged spleen, diarrhoea, or abdominal symptoms. On the fifth day of the disease the Widal reaction became positive in a dilution of 1:50. This child died on the sixth day of the disease, with increasing signs of pneumonia and a positive Widal reaction of 1:350. Many of these cases of typhoid fever in older children become comatose after the first week. Such a case was recently admitted to my wards. The onset was with headache and fever. There was no vomiting, epistaxis, or chill. The child became unconscious, with a temperature of  $106^{\circ}$  F. ( $41.1^{\circ}$  C.), rigidity of the muscles of the neck, increased reflexes, ankle-clonus, Kernig's symptom, and enlarged spleen. This case gave a positive reaction to the Widal test, and

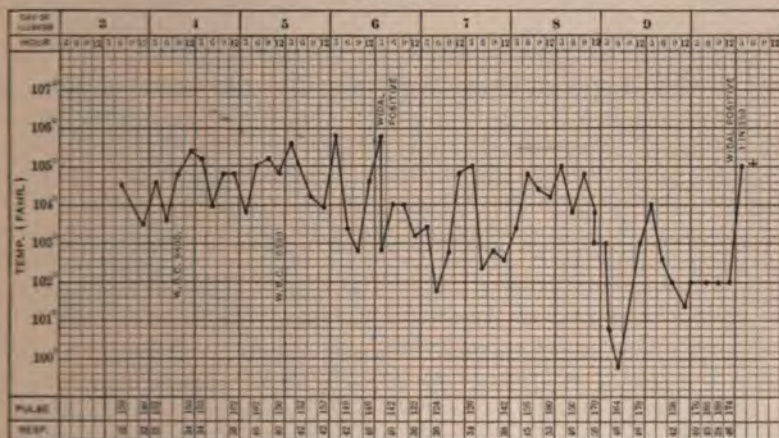


lumbar puncture failed to reveal anything characteristic in the fluid withdrawn.

The invasion is not characteristic in infants. In exceptional cases (Blackader) a convulsion is the first symptom noted. In some cases there may be a simple continued fever with diarrhoea, without other symptoms. In a case reported by Crozer Griffith the roseola and the enlarged spleen were present.

The subsequent history of a case varies with the character of the infection. In the forms which have a slow, gradual onset the children remain for a time in good physical condition. During the first week the sensorium is clear, the tongue coated, and the face of good color; the spleen may be readily palpable, the roseola appears, and there may be diarrhoea or constipation. In some cases the iliac

FIG. 38.



Typhoid fever which began as a lobar pneumonia in a girl four years of age. Consolidation of the lower lobe of the left lung; death on the tenth day of the disease.

tenderness is marked; in others absent. It may not be possible to determine the presence of ileocecal tenderness in young children. The symptoms after the first week may be augmented by delirium at night; in older children this delirium, which has much the same character as in the adult, is also present during the day. Children from five to seven years of age are more likely to have the quiet form of delirium, while older children are noisy and try to get out of bed.

The course of pneumonic cases is noteworthy. Resolution is tardy in those cases which recover. To the symptoms of pneumonia are added after a time those of typhoid fever—roseola and enlarged spleen. The temperature-curve is not characteristic, and resembles that of the sustained remittent type (Fig. 38). In some cases pleurisy may be present.

In the newly born infant to whom the fever has been conveyed in utero the picture of the disease is unlike that seen in older infants and children. The symptoms resemble those of sepsis of the newborn. Thus in the case published by Blumer the first symptom of the disease was an uncontrollable hemorrhage from the vagina. Before death this was supplemented by hemorrhages into the skin and from the gums.

The cases of typhoid fever in infancy thus far recorded by Morse, Crozer Griffith, Blackader, and the author, may be divided into two classes: those in which there is a mild diarrhoea with distention of the abdomen, roseola, and enlarged spleen; and those which present cerebral symptoms. The latter develop coma, have a distended abdomen, rose spots, and enlarged spleen. In both forms there are severe and mild types. Cases in which the temperature rarely rises above  $104^{\circ}$  F. ( $40^{\circ}$  C.) recover, while those with a higher temperature may be fatal.

**Individual Symptoms.**—**ROSEOLA.**—In children, as in the adult, the roseolar papules are seldom absent. In some cases their number is large, while in others they are few and widely scattered over the surface. They may appear in successive crops, and reappear in a relapse. Occasionally the roseola is preceded by a diffuse erythema closely resembling the scarlet fever eruption. The roseola may, as in the adult, appear on the third, fifth, or tenth day, and may even be delayed until the end of the second week, after which it gradually fades, leaving a pigmented area. The eruption is sometimes so profuse as to resemble the eruption of typhus. It may be profuse in cases in which the cerebral symptoms are very marked. I have seen typhoid fever with severe cerebral symptoms, but with an eruption very sparse or entirely absent at the height of the disease. In severe delirious cases, hemorrhagic areas appear on the bony prominences of the shoulders and extremities. Petechiae are common. In protracted cases extensive purpuric areas appear on the abdomen. These hemorrhagic cases are not necessarily fatal.

The **ENLARGED SPLEEN** is one of the most common physical signs. At the outset of the disease it is not always easy to palpate the spleen. This is especially true of younger children. The enlarged spleen is present not only in older children, but also in cases of fetal typhoid fever. I have seen the enlargement persist for weeks after convalescence. In one case the spleen could be distinctly felt below the border of the ribs for a long time after recovery.

In some forms of relapse the spleen enlarges after having diminished to the normal size. Cases in which the spleen remains enlarged a long time are likely to have slight rises of temperature of short duration. Typical relapses without enlargement of the spleen may occur. The fact that the spleen continues enlarged after



the temperature has become normal does not always indicate the approach of a relapse.

**Temperature.**—An elevation of temperature in young children is usually not observed during the first eight days. Children rarely complain of slight malaise, and a rise of a degree or even more above the normal may escape notice; as a result, the impression is prevalent that the temperature during the first week does not follow the typical curve. The cases which I have observed sufficiently early, and which were not complicated with pneumonia, showed during the first week the gradual rise seen in the adult (Fig. 39). This gradual daily rise of temperature is also seen in relapses. On each day the temperature at its highest point is higher than on the previous day. After the first week the temperature is likely to show a remittent curve with a sustained maximum point. After the second week the temperature may remit, gradually falling, or intermit; frequently it remains high for weeks, with daily remissions. By the end of the second week it reaches  $104^{\circ}$  to  $105^{\circ}$  F. ( $40^{\circ}$  to  $40.5^{\circ}$  C.) at its highest. In the course of the third, fourth, and fifth weeks it may range a degree lower, with remissions to  $101^{\circ}$  F. ( $38.3^{\circ}$  C.), not reaching the normal. If the case is protracted, the temperature may persist into the sixth week, running up as high as  $106^{\circ}$  F. ( $41.1^{\circ}$  C.), falling fully five degrees twice daily. In one case the temperature did not become normal until the eighth week. Even at this late period there may be relapses. In many cases the temperature falls to the normal after six or seven weeks, or becomes subnormal, and then after an interval of a few days or a week rises

Typhoid fever in a boy five years of age, observed from the outset. Widal reaction positive on the fifteenth day.

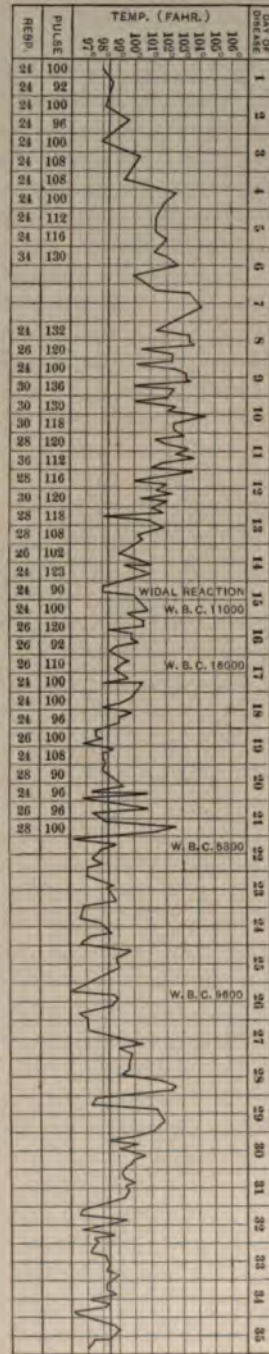
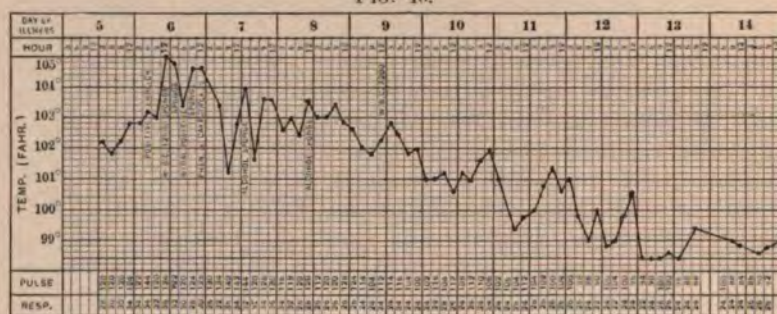


Fig. 39.

and fluctuates a degree or more above the normal. This continues for a few days, the temperature remitting to the normal or near the normal. These post-typhoidal fluctuations are sometimes mistaken for relapses. They are rather to be attributed to inanition, or are the result of slight absorption from the gut. In a large number of cases the first sign of convalescence is a subnormal temperature. On the other hand, the temperature may be subnormal for a week or more and relapse follow (Fig. 40).

It may be said that as a rule the first week of typhoid fever in children shows a gradual rise of temperature. The subsequent temperature is sustained, remitting two or more times daily. This curve may last one, two, or more weeks. In other words, there is no characteristic temperature-curve. In relapses the temperature rises gradually from day to day. Among the causes which may give rise to a slight temporary elevation of temperature is constipation. A

FIG. 40.



Typhoid fever of short duration in a boy six years of age.

lobar pneumonia or a bronchopneumonia will cause a persistence of the high temperature, as will also other conditions, such as otitis.

The inverted type of temperature-curve is described by Hensch. The morning temperature is higher than the evening, or there may be a rise at 3 A. M. or 6 A. M., a fall in the forenoon, with a rise again at noon, and a fall toward evening. Such a curve may be followed within a day or two by the usual fall in the morning and rise toward evening. These fluctuations occur at the height and at the decline of the disease.

**Hemorrhages** from the bowel are not so common in children as in the adult. I have seen persistent hemorrhages in only 4 out of 84 cases. In one case I have met post-typhoidal ulcerative colitis. The bowels may be constipated, normal, or diarrhoeal. The number of stools varies. In the majority of cases diarrhoea is absent. In some the temperature in convalescence may rise a degree or more for a day or two. In these cases there may be fecal accumulation due to incomplete evacuation of the gut.



Sensitiveness in the ileocaecal region is very difficult to determine in young children. In older children it is sometimes marked, and indicates ulcerative processes in that region or in the neighborhood of the appendix. Perforation is rare in children. In the case of a boy of nine years the pain, collapse, elevation of temperature, and rise of the pulse above 120 indicated perforation, but operation revealed no perforation. Recovery resulted. In another case, also a boy of nine years, there was great ileocaecal tenderness, with a sudden rise of temperature and local rigidity, but the pulse remained below 120. There was an increase in the number of leucocytes to 10,000. There may have been ulceration and localized peritoneal reaction in this case, but no perforation.

**Otitis** is not uncommon. I have seen several cases.

I observed **parotitis** in only one case.

The **tongue** of children with typhoid fever resembles that of the adult. It is at first coated, and is protruded in a tremulous manner; subsequently the epithelium is thrown off and the papillae become prominent. In some cases the tongue resembles the so-called strawberry tongue seen in scarlet fever. At the height of the disease it may become dry and fissured, and sordes may collect on the teeth. The lips become fissured and bleed easily.

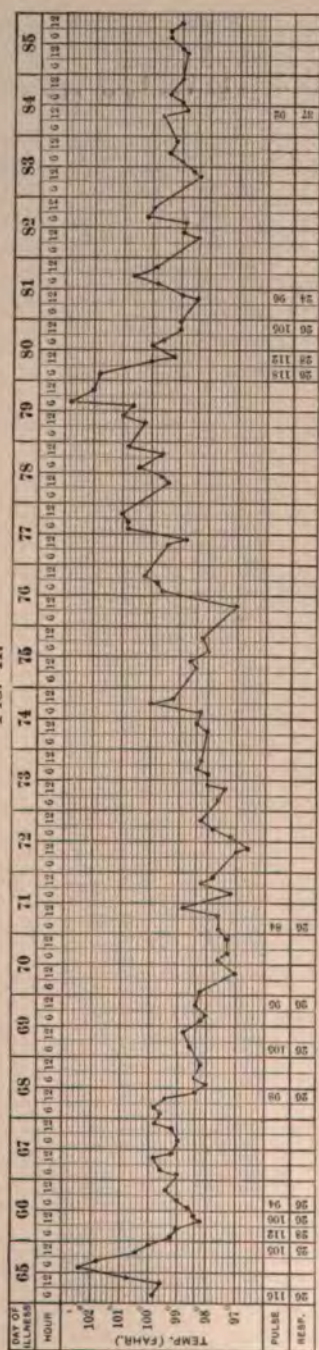
The **nervous symptoms** of older children resemble those of the adult. With younger children sopor is the rule and delirium is infrequent. Melancholia or depression occasionally is met with in convalescence, usually in girls of hysterical temperament.

**The Heart.**—In a recent epidemic of typhoid many cases showed systolic apex-murmurs. These murmurs were loudest over the base, close to the sternum, or over the pulmonary orifice. Such murmurs are myocarditic. In one case there was a loud musical systolic murmur heard over the apex of the heart. It was also heard at the base of the heart. The murmur appeared early in the third week. There was also a pleuropericardial friction-sound. Post-mortem examination revealed myocarditis and pleuropericardial adhesion.

**The Lungs.**—The occurrence of lobar or lobular pneumonia late in the course of typhoid is serious. At this time the patient's powers of resistance are greatly diminished. Especially grave are the cases which show a sustained high temperature for two or three weeks, and then develop pneumonia. If with the pneumonia there are extensive hemorrhages under the skin at the situation of the bony prominences, the outlook is grave. In such a case I have seen a pneumonia involve the whole lobe of the lung in consolidation within a few hours.

**The Blood.**—In children, as in the adult, the number of red blood-cells diminishes, and reaches the lowest point at the end of the febrile period. The hæmoglobin also is diminished. The leucocytes are diminished from the outset until convalescence, but increase after

FIG. 41.



Typhoid fever; temperature normal on the twenty-eighth day of the disease. Relapse on the fifty-third day. Recrudescence of the temperature after the sixty-sixth day without a true relapse.

it is established. In one of my cases their number fell to 3500, and then rose to 12,400. In a case complicated with extensive ulceration in the gut and bronchopneumonia they numbered 30,000. In fatal cases complicated with lobar pneumonia I have found them as low as 4500. According to Thayer, the polynuclear neutrophils steadily diminish as convalescence approaches, while the mononuclear leucocytes and eosinophiles increase. With the establishment of convalescence blood conditions return to the normal.

**Relapses.**—A relapse is a gradually ascending temperature-curve extending over a week or longer after the temperature has been normal for a time (Fig. 41). A relapse was noted in 7 of 46 cases of my last series. In all, it was mild and no serious results followed. On the other hand, a prolonged low febrile curve causes great emaciation in children. Importance has been attached to the condition of the spleen in these cases. The percentage of relapses varies with the nature of the prevailing epidemic. Blackader records 15 relapses in 100 cases, and Henoch 44 in 375 cases. Apparently relapses occur independently of the mode of treatment and diet.

#### Complications and Sequelæ.

—**Skin.**—Subcutaneous abscesses may occur, and onychia is common. Erysipelas and parotitis are rare. Œdema may be confined to the scrotum, or during defervescence the whole surface of the body may be œdematous,



In a case of scrotal oedema coming under my observation there were no casts or albumin in the urine; the leucocytes were diminished. Henoch attributes oedema to cardiac weakness rather than to nephritis.

**Diphtheria** is a very serious complication. I have observed it in 2 out of 84 cases.

**Lungs.**—Bronchitis is a frequent complication. In the later stages of the disease in younger children it is likely to develop into bronchopneumonia, especially in cases in which the course of the disease is protracted. Pneumonia may occur. Gangrene of the lung is mentioned by Henoch as a rare complication.

**Arthritis** is uncommon. Usually only one joint is affected. It occurs in the post-typhoidal period and runs a favorable course.

Among the **nervous symptoms** which complicate or follow typhoid fever are aphasia, amblyopia, ataxia of the lower extremities, paralysees of various sets of muscles, double ptosis, and hemiplegia. In hysterical children there may be a post-typhoidal melancholia. In others stupidity may persist for a time. Recovery usually takes place in all forms of paralysis, aphasia, or melancholia. The paralysees are possibly due to a neuritis of toxic origin, as is the case with the other infectious diseases. Hemiplegia occurs only as a result of embolism (Henoch). I have met cases of ataxia and marked melancholia. The children made an excellent recovery. In one case, a boy of four years, catalepsy was present for a period of five weeks after the temperature had become normal.

The **duration** of typhoid fever varies within wide limits. Henoch in his tabulation of more than two hundred cases shows that the shortest duration was seven to nine days; the longest, seventy days. Relapses and conditions of inanition are not included. In my cases the duration varied widely if the recurrent rises of temperature were taken into account. In 30 cases the shortest duration of the fever, exclusive of relapses, was ten days; the longest, forty-six days; the average duration, twenty-one and one-third days. Including the relapse, which continued forty-eight days, one case lasted seventy-five days, the original fever having lasted twenty-six days.

**Diagnosis.**—Enough has been said to show that the diagnosis of typhoid fever in infancy and childhood is at times very difficult. With young children enteritis, pneumonia, meningitis, and even appendicitis may simulate typhoid fever in their onset. Cases which begin as a pneumonia are especially difficult of diagnosis. The cerebral forms of typhoid fever may closely resemble meningitis. The history is very important. The onset of typhoid fever is gradual, the cerebral symptoms increasing in intensity as the disease progresses. An enlarged spleen and a few roseolar papules will be of service in making a diagnosis, but, on the other hand, an enlarged

spleen is common to many conditions of infancy and childhood. In the most puzzling cases, such as those simulating enteritis of non-typhoidal nature, the roseola may at the outset be absent.

In a doubtful case the Widal blood-test should be made daily to clear up the diagnosis. In many cases this reaction is the only clue to the condition. During the prevalence of an epidemic every case of pneumonia or doubtful meningitis or enteritis should be subjected to this test.

The **Widal agglutination reaction** is of greater utility in making a positive diagnosis of typhoid fever in children than in adults. The fact that an enlarged spleen may be due to various causes, such as rickets, the occurrence of fevers of a remittent or continued type, possibly due to otitis, enteritis, pneumonia, and the prevalence of diarrhoea of all kinds in infants and children, tend to make the Widal test of inestimable value.

In a paper based on 84 of my cases of typhoid fever in infants and children, Gershel found the reaction positive in 81. Three hundred and twenty-nine examinations in all were made. Thirty-six were positive on the first test, and forty-five on repeated tests. The reaction appeared in 5 cases on the fifth day, in 3 cases on the sixth day, and in 3 on the seventh day. In other words, 13 per cent. of the tests were positive at the end of the seventh day, 63 per cent. on the fifteenth day, and 89 per cent. on the twenty-fifth day of the disease. The reaction was negative in only 3 cases which gave the clinical symptoms of typhoid fever. These figures correspond to those obtained by Blackader in a smaller number of cases. A negative reaction is of no significance as excluding typhoid fever, whereas a positive reaction is absolutely pathognomonic of the disease. Though 115 of my fever cases were examined by this test for the presence of typhoid fever, exclusive of the above 84 cases, the reaction was obtained in no case in which typhoid was not present. In a few cases the reaction was not obtained until the close of the disease, when the temperature had been normal for some days. In another case of a child of three years, the reaction was not obtained until a relapse had occurred. In the case of a boy of seven years the reaction was not obtained until the third week. It was not taken again until the onset of a relapse in the fifth week, and was negative. It subsequently became positive at the termination of the relapse. This proves that a negative test is of little significance in excluding the possibility of typhoid fever unless the examinations extend over a long period. The presence of the reaction in typhoid fevers which begin with a pneumonia is of interest. In one of these cases, fatal on the eighth day, in a child four years of age, the reaction was negative until the fourth day of the pneumonia. It became positive in an attenuation of 1 : 350 just before the exitus lethalis.

**The Ehrlich Diazo Reaction in the Urine.**—Thirty-three cases were



examined with reference to this reaction. The fifth day was the earliest day on which it was obtained. In the majority of cases the reaction was present from the seventh to the tenth day of the disease. The latest appearance was on the forty-seventh day from the outset of the disease. The reaction was absent in 15 per cent. of the cases. In all of the cases in which the Ehrlich reaction was obtained the Widal test was positive, and appeared in the first two weeks of the disease. The reaction may be present, as it was in one case, on the fifteenth day, and be absent on the next. The diazo reaction may appear before the Widal reaction, but in some cases the contrary is true. In conclusion, it may be said that in the presence of symptoms and signs of typhoid fever the diazo reaction is an aid to diagnosis, although not pathognomonic of the disease.

Of the clinical signs pointing to typhoid fever, the character of fever aids us but little. In the third week it may become intermittent, thus simulating malarial fever. In other cases the fever may be sustained with daily remissions until the fifth week. Typhoid fever with great ileocaecal tenderness and pain may closely simulate appendicitis. In a recent case published by Berg, operated upon for appendicitis, the operation revealed that the patient was suffering from a perforation of the appendix due to an ulcer of typhoidal origin. A continued fever of longer duration than a week, a tremulous tongue, facies, a pulse below 120, an enlarged spleen, and a few roseolar spots, will aid in the diagnosis. The diagnosis of perforation of the gut is not always simple in children. In these subjects tympanites is not uncommon, aside from the presence of peritonitis. I have cited a case in which all signs pointed to perforation, and yet operation revealed nothing. The classical signs of perforation are those of collapse, a sudden fall of the temperature, and a rise of the pulse above its normal frequency, an increase of the leucocytes, and the presence of tympanites increasing until it causes a disappearance of the liver dulness. No one of these symptoms is absolutely pathognomonic. In many cases we shall be compelled to draw conclusions from the general history of the case.

The diagnosis of typhoid fever must, therefore, be confirmed by the Widal reaction, except in a small percentage of cases. The presence of roseola, enlarged spleen, facies, tremulous tongue, diarrhoea, and continued remittent fever are the clinical symptoms which should lead the physician to apply the test.

The **prognosis** of typhoid fever in infancy and childhood is, as a rule, good. The mortality varies with the severity of the infection and the character of the epidemic. If the infection is severe, the complications will militate against recovery. Henoeh, in 375 cases had a mortality of 14 per cent.; Blackader, in 100 cases lost only 1; Crozer Griffith had a mortality of 3 per cent., and in my last series of 46 cases the mortality was 8.7 per cent.

The **treatment** of mild cases of typhoid fever is purely symptomatic. There is little need for the administration of medicines. On the other hand, the severer cases are difficult to manage. This is especially true in the treatment of children, to whom it is not always possible to apply methods adopted with the adult. In cases in which delirium is present night and day bromides in large doses are efficacious. With older children they may prove useless, and morphine may then be necessary to meet the exigencies of the case.

In the vast majority of cases milk forms the basis of the diet. If there is progressive emaciation, one, two, or three raw eggs should be added to the milk daily. It is well in protracted cases not to wait too long for a complete drop of temperature before resorting to other foods than milk. This is especially true of cases extending over a period of seven or eight weeks, in which there is always a rise of temperature of half a degree or a degree above the normal for a few days, with a drop again to the normal or subnormal. In these cases there is a form of inanition fever, post-typhoidal in nature. Solid food should not be withheld too long lest the emaciation become extreme. After the fifth week we may in most cases allow the patient gruels containing cereals. After the temperature has fallen to the normal and remained there for four or five days, it is safe to return gradually to a full diet. It is doubtful if relapses occur as a result of too early feeding if this method is followed. In comatose states resort may be had to forced feeding.

**Alcohol** is not needed in mild cases. It is given in cases in which the pulse is weak and the temperature high. Delirium is no contraindication to its use, as it is in other affections.

The **heart** is stimulated by digitalis, strychnine, or camphor. If the heart has shown slight dilatation with a murmur developing in the course of the disease, the patient should not be allowed out of bed too soon for fear that unfavorable symptoms may result.

The temperature is controlled by hydrotherapy. The patient is placed in a bath at 100° F. (37.7° C.), and the temperature of the water gradually reduced to 85° F. (29.4° C.). With older children the temperature may be lowered still further. Children do not bear the classical Brand bath treatment well. The plunge bath is given three or four times daily whenever the temperature is 103° F. (39.4° C.) or more. Should the child struggle very much against the administration of the bath, it is wiser to forego it and substitute sponging. If the sponging is not followed by good reaction, the use of water should be abandoned. In exceptional cases of delirium a bath once or twice daily at 105° F. (40.5° C.) has a quieting effect.

**Hemorrhages** from the bowel are not frequent in children. They may occur early or late in the disease. In the latter case they must be differentiated from hemorrhage due to enterocolitis of a post-



typhoidal character. In hemorrhage due to typhoidal ulcer an ice-bag is applied to the abdomen, and small doses of opium, preferably the deodorized tincture, are administered to control peristalsis. Ergot and digitalis are given internally in order to contract the bloodvessels if possible. Enemata should not be given. If the hemorrhage becomes excessive, it is proper to give hot saline enemata, and to infuse normal saline solution under the skin or into the veins.

**Enteritis** of an ulcerative or pseudomembranous character occurring as a complication of typhoid fever is treated in the same manner as the primary affection.

**Perforation** should be treated on surgical principles. As with adults, those perforations which occur late in the disease, when the patient is in an exhausted and emaciated condition, give a less favorable prognosis than those which occur early. In cases in which the diagnosis is doubtful it is best not to operate.

**Constipation.**—In most cases of typhoid fever one enema a day will remove accumulated feces from the lower bowel. If the bowel contents are streaked with blood, enemata should be discontinued. In cases in which there is a slight rise of temperature during convalescence without apparent cause, grains  $v$  (0.3) hydrarg. cum creta should be given. Tympanites is treated as in the adult subject. The evacuations should be mixed with an equal volume of a solution of carbolic acid (1:20) as soon as passed. The hands of the nurse should be thoroughly cleansed after each movement. The patient's hands are cleansed daily, in order to avoid auto-infection.

## MALARIAL FEVER.

(*Paludism ; Malaria ; Intermittent Fever.*)

Malarial fever is an acute infectious disease due to the inoculation of the individual with the *Plasmodium malarie*. It is common in infants and young children, and is believed to occur in utero. Crandall has reported a case in which symptoms developed eighteen hours after birth, and in which the plasmodium was found in the blood of the infant. Those who, like Moncorvo of Brazil, have opportunities to observe malarial fever in young infants and children, find the greatest frequency under two years. The author has not met paludism as frequently in the nursing infant as in older children. The reason for this must lie in the fact that young infants are more protected from infection with veils, etc., than older children. One attack does not confer immunity to subsequent attacks; on the contrary, infants and children once the subject of paludal poisoning seem particularly liable to reinfection and relapses.

The period of incubation varies from a few hours to weeks. In

the tertian type it is believed to be from seven to fourteen days. In one of my cases the first chill appeared eleven days after the patient had left the malarious district.

**Etiology.**—The essential cause of malarial fever is the same in infants and children as in the adult. It is an inoculation fever, and is conveyed to the human subject by a certain species of mosquito (*Anopheles*). The poison exists in the neighborhood of swamps and stagnant waters.

**The Parasite.**—The plasmodium or protozoa of malaria circulates in the blood of infants and children, undergoing its cycle and sporulation in the same manner as in the adult. In one series of cases in infants and children that I studied, the tertian was the most prevalent form of parasite. These cases occurred in New York City and its vicinity. This has been the experience of other New York City observers. One may assume that the blood will, as a rule, contain the parasite prevalent in a given locality. Several forms of parasites may exist in the blood of the same child, or there may be several generations of the same plasmodium. These may mature at different times, giving various types of fever in the same subject. In a tertian case, the fever may thus become quotidian, a second set of parasites causing a distinct chill and fever (paroxysm) on the day when the first generation is quiescent. We may have, as Mannaberg and others pointed out, simple and double tertians and quartans. But no combination of quartan parasites can simulate the simple tertian type. I have seen very few cases of quartan in children. They are uncommon in New York City, but I have seen preparations of the quartan type which were found in the blood of children in the Southern States. As in adults, tertian paroxysms may occur every day, caused by two sets of parasites which mature at about the same time daily, or one set matures at a different hour than the set of the following day. In such a case paroxysms would occur at the same hour only every other day. Many children have a distinct severe paroxysm only every other day, but on the intervening day a careful examination will detect a very low fever. This is probably due to a set of parasites which mature without producing marked chill or fever (abortive).

**The Blood.**—In recent tertian I have found young spores in abundance in the blood a few hours after the chill. In some specimens the spores were free. Between paroxysms in tertian cases the blood contains colorless oval plasmodia—the fully developed body—leucocytes having rods and pigment-granules and rarely, small round forms with flagellæ (Koplik). In stained specimens (methyl-blue) young native forms are found in all stages up to fully developed protozoa. The red blood-cell containing the parasite is distinctly enlarged. I have found in the stained specimen as in the unstained ones, the sporula in free groups, bodies with flagellæ, and erythro-



cytes with stained granules. The half-moons are also found in chronic cases. The blood contains free granules, and peculiar shrunken, brassy-colored, red blood-cells. Monti found the specific gravity of the blood to be increased.

**Morbid Anatomy.**—Post-mortem examinations in cases of malarial fever in infants and children are exceedingly rare. Opportunity may be afforded when death occurs as the result of accident or of some other disease. Monti states that in fatal cases the spleen is enlarged; the capsule is tense, and in places shows rupture. The pulp is dark red owing to pigment deposit (melanin). Old spleens show a disappearance of melanin and a deposit of yellow ochre pigment along the trabeculae. In chronic cases the connective tissue is increased, the liver is enlarged, and there is atrophy of the liver-cells. The parasites are found in the blood. The endothelium of the bloodvessels contains yellow and brown pigment. In exceptional cases there are melanin deposits. In acute cases the bone-marrow is the seat of melanin deposit; later this disappears, and the marrow is found to be yellow and fatty. The brain cortex in severe cases shows pigment deposit; sometimes there are thromboses and hemorrhages.

**Symptoms.**—Children living in malarious districts do not always manifest malarial poisoning by having paroxysms of chills and fever. The disease is masked under the form of a progressive anæmia, with accompanying enlargement of the spleen. These patients develop symptoms in from a few days to a few weeks after leaving the malarious region. The period of incubation is thus distinctly indicated.

The onset of a paroxysm is usually marked by the appearance of chills. In young infants a distinct chill is not always present. They become cold and blue at a certain time each day. In older children the paroxysm is indicated by headache and a feeling of lassitude, which comes on at a certain time each day, or by a distinct chill. In exceptional cases eclampsia or vomiting may usher in a paroxysm. In other cases there is no eclampsia, but the hands become cold, there is a feeling of faintness, and the child complains of being ill. Meanwhile there is a rise of temperature, during which there are muscular tremors of the extremities and a peculiar upward rolling of the eyes, indicating an impending convulsive seizure. The chill may occur during sleep. In one case the mother noticed that the child (three years of age) became pale during sleep, the hands and extremities became cool, and the pulse rapid. The febrile movement following the chill may be very slight, scarcely half a degree above the normal. In such cases the chill is not marked or is scarcely noticeable. This occurs in double tertian, in which one paroxysm is abortive. In most cases the fever is very high at first—so high that it is characteristic. A tempera-

ture of 106.5° F. (41.3° C.) is not uncommon, and is well borne. As a rule, the fever has a distinctly intermittent type. The temperature may rise after the initial chill and remain high for days, and then fall to the normal. In the simple form the fever lasts from four to twelve hours, and is followed by a critical perspiration, during which the temperature rapidly falls to the normal. In some cases the children appear free from symptoms in the interval between the paroxysms. Others suffer from headaches and a feeling of lassitude, and in infants there are gastric and intestinal disturbances. In protracted cases a distinct anæmia develops, with progressive enlargement of the spleen. Neuralgia of the peripheral nerves has been noted in older children.

During a paroxysm Monti noted polyuria, which persisted until the following day.

The spleen enlarges rapidly, and in a short time may be felt as low down as the umbilicus. I have found the spleen markedly enlarged; in one case the organ was not palpable below the ribs, although a slight enlargement could be detected on percussion.

The liver may be enlarged in chronic cases.

In subacute forms chills are not present, but there is an irregular febrile movement, with progressive anæmia and splenic enlargement.

**Repeated Attacks or Relapses.**—Children, as well as adults, may have repeated attacks of malarial fever. As a rule, however, these so-called independent attacks in children are relapses, due either to inefficient treatment or to the development of a new series of parasites. Infants may have relapses. I have treated such cases until all anæmia and signs of active malarial poisoning had disappeared, and then administered arsenic for months, only to find a return of the symptoms after an interval of months.

The **diagnosis** of malarial fever is based upon an examination of the blood. If a child suffers from pronounced anæmia, malaise, pains in the limbs, and enlarged spleen the blood should be carefully examined. Expert knowledge is always necessary for a definite diagnosis. It is surprising to note the large number of cases beginning with chills and presenting an intermittent fever curve and enlarged spleen, diagnosed as malarious, in which parasites cannot be detected in the blood. Many septic and inflammatory processes in infants and children simulate malaria. Rachitis, syphilis, gastro-enteric catarrh, otitis, pneumonia, typhoid fever with relapses, have all been mistaken for malarial fever. The diagnosis rests on an examination of the blood in all cases in which chills and fever or any of the symptoms described coexist with enlargement of the spleen.

Quinine should not be administered until the blood has been very carefully examined. In other words, malaria should be diagnosed or excluded before resorting to this remedy, which was formerly much in vogue as a diagnostic test. Its use before diagnosis can



only result in uncertainty, since there are rises in temperature, not due to the paludism, which may be influenced by quinine. A very high temperature of an intermittent type, in connection with other physical signs, should cause the physician to consider the possibility of paludal poisoning.

I have not seen cases of the pernicious type. They occur in the Southern States.

Acker has recently published 2 cases of malarial fever in children, in which there were the initial cerebral symptoms of coma and convulsions. Coma in one case came on in paroxysms. In the interval the child was rational. The æstivo-autumnal parasite (pernicious) was found in the blood.

The **prognosis** of malarial fever in New York City is very good. With proper treatment the patient should recover. I have never met a fatal case. They occur in districts in which the pernicious type of the disease is prevalent.

**Treatment.**—If possible, the patient should be removed from the malarious district. The remedies employed in all cases are quinine and arsenic, or their derivatives.

According to Golgi, quinine should be given before the paroxysm, and also in the intervals. The action of the drug is exerted directly upon the plasmodium. At this time segmentation of the parasite takes place in the blood, and most of the young parasites are free in the plasma. They then respond most quickly to quinine. Large doses should be given to infants and children, in order that the infection may be destroyed quickly and completely. The soluble bisulphate and muriate are suitable preparations. To an infant under one year of age grains  $\text{ij}$  (0.1) are given in a dose, repeated three times a day, the last dose being given from three to five hours before a paroxysm. To children between two and five years of age grains  $\text{ij}$  to  $\text{v}$  (0.2 to 0.3) are given in the same manner. Some infants take quinine readily when it is suspended in powder form in milk or water; others are given a piece of chocolate, and when the surface of the mouth is coated with the candy the drug is administered. The syrup of yerba santa is also a good menstruum. In cases in which children cannot take quinine by mouth, Jacobi advises giving it per rectum, dissolving the drug in a solution of tartaric acid. I have never been forced to use subcutaneous injections of quinine, as the pernicious form of malaria in which this mode of therapy is principally resorted to is not prevalent in New York City.

Infants and children with chronic or subacute forms of malaria are likely to be constipated. Under these conditions I have found calomel more efficient in clearing the gut than castor oil.

After the quinine treatment has been continued for some time the spleen will be observed to diminish in size and the paroxysms

to disappear. If the anæmia persists, it is well, after diminishing the frequency of the dosage of quinine, to combine it with small doses of Fowler's solution. The arsenic must occasionally be temporarily discontinued, or the functions of the stomach will become deranged. Warburg's tincture does not seem to be very efficacious with children under five years of age, nor with older children, unless given in very large doses. Children do not develop cinchonism as quickly as adults, and the quinine may therefore be continued for a long time. Treatment should not be suspended until the spleen is no longer palpable and the anæmia has disappeared. Quinine should then be continued in small doses at regular intervals.

The preparations of cinchona, such as cinchonidia, cinchonidin, chinidin, etc., are not reliable. The following is Baccelli's formula for the subcutaneous use of quinine in pernicious intermittent fever :

Quinin. muriat. . . . .	15 grs. (1.0).
Natrium chlorat. . . . .	1 gr. (0.06).
Aq. destillat. . . . .	3iiss (10.0).

## INFLUENZA.

(*La Grippe ; Acute Catarrhal Fever.*)

Influenza is a specific infectious disease chiefly affecting the mucous membranes. It is highly contagious, although all individuals exposed do not contract the disease. It occurs in the form of pandemics in which whole communities are affected. This pandemic form occurs less frequently in children than adults, and is of interest to the physician only when an epidemic prevails. The endemic form of influenza affects children more frequently than adults, and is the form which will be described, although in its symptoms it closely resembles the epidemic form. The endemic form may occur at any season of the year. In large cities influenza is always present (endemic), and appears to be more prevalent after rapid changes from lower to higher temperatures. Rapid fluctuations in the humidity of the atmosphere in winter also favor the development of the germs of this disease. In New York City, midwinter and spring are the seasons when outbreaks of this affection occur.

**Age.**—Influenza may affect the newly born infant. A case of this kind is reported by Townsend in the *Transactions of the American Pediatric Society*. The disease is most frequent between the ages of six months and five years. The younger the child, the more severe the affection.

**Mode of Infection.**—Individuals are infected by coming into contact with others suffering with the disease. The germ is contained in the sputum and the nasal secretions ; therefore poorly



ventilated rooms and public conveyances favor the transmission of the disease. Parents may transmit it to their children in the act of kissing, and wet-nurses who have la grippe are likely to infect the infant at the breast.

**Etiology.**—The epidemic form of influenza has been studied by Pfeiffer and Kitasato. Pfeiffer isolated a bacillus from the bronchial mucous membrane, trachea, and lungs. This bacillus, which is now believed to be the essential cause of epidemic influenza, is exceedingly small, and two or three times as long as it is broad. It has rounded extremities, occurs in pairs and chains, does not stain by Gram's method, and in influenza, pneumonia, and encephalitis is found in enormous numbers in the lungs. It is called the *Bacillus influenzae*. It is still an open question whether it occurs in the blood. Although this bacillus has been found in sporadic cases of endemic influenza, competent observers, Luzzato among the latest, have found that in a large number of endemic cases of influenza the Pfeiffer bacillus is absent. In its place is found the Fränkel diplococcus. This is thought to be the essential cause of an important group of cases of endemic and sporadic influenza in children—the so-called pneumococcus grippe. Predisposing elements in the etiology of endemic influenza are exposure to cold and a diminution of the strength of the individual. One attack does not protect the individual from subsequent attacks.

**Incubation.**—Influenza is believed to have an incubation period of from twelve hours to three days. Endemic influenza occurs frequently in large cities and at times local epidemics of the disease are seen.

**Morbid Anatomy.**—Inasmuch as influenza is rarely fatal, the pathological anatomy is imperfectly formulated. In fatal cases a general inflammatory condition of the mucous membrane of the nasal passages, and of the larynx and trachea, is found. The surface of the lining membrane of the bronchi is reddened, covered with mucus, and the membrane itself is infiltrated with small round cells. There may be a diffuse inflammation of the smaller bronchi, with peribronchitis and inflammatory reaction. Areas of bronchopneumonia or lobar pneumonia are found in the lungs. The heart is dilated and the seat of myocarditis. There may be endocarditis and the kidneys may present an acute nephritis. The pleurae are inflamed, and there may be serous or serofibrinous pleurisy or empyema.

Among the other lesions are those due to the complications, otitis, meningitis, inflammation of the gastro-intestinal tract, and cerebrospinal meningitis.

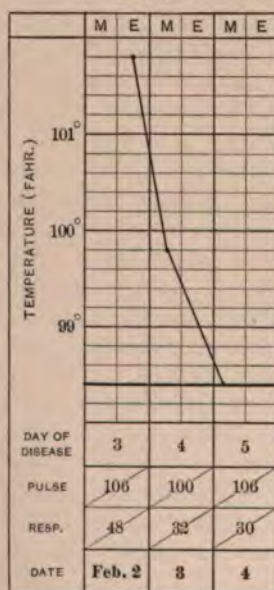
**Symptoms.**—It has been customary to divide the symptomatology of endemic influenza as it occurs in children into clinical forms. According to my experience, there is no sharp dividing-

line between the various forms of endemic influenza as seen in children. The gastro-intestinal, nervous, and pneumonic forms are frequently present in the same patient. Endemic grippe as it occurs in children in New York City will be described, the epidemic or pandemic form being ignored.

The most frequent form is the catarrhal of an acute and even subacute type. The infant or child may at the outset have a chill. Most frequently there is vomiting, and also fever, and pains in the head and limbs. There is a coryza, and in many cases a croupy, barking cough. The eyes are injected, the face is red and flushed, and the child presents an appearance resembling that of the first stage of measles. The mucous membrane of the throat is deeply injected and the tonsils inflamed and enlarged. The temperature is elevated; in fact, at the outset it is as high in this disease as in malarial fever,  $106.5^{\circ}$  F. ( $41.3^{\circ}$  C.). The cough is sometimes incessant. The irritation in the throat is extreme, and

vomiting after the coughing paroxysm may lead the physician to believe that he is dealing with whooping-cough. In young infants these symptoms may last for a day or two, during which the movements may become green and even diarrhoeal. This diarrhoea is sometimes so severe as to be a prominent feature of the disease. The prostration both in infants and children is marked. After two or three days the catarrhal condition of the upper air-passages subsides, and the patient develops symptoms of an acute bronchitis of a severe type. These forms of grippal bronchitis have at the outset a high febrile curve (Fig. 42), and a fever persisting for days. The bronchitis affects the smallest bronchi. There may develop a bronchopneumonia in small areas. In other cases the bronchitis passes suddenly into a pneumonia without a preceding chill. The pneumonia of la grippe may be lobular or lobar in type. In the vast majority of

FIG. 42.



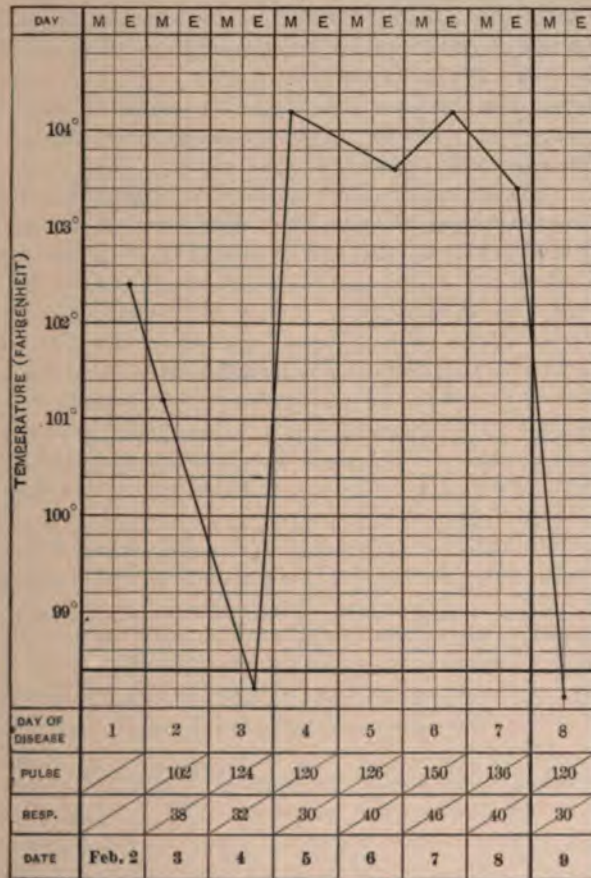
Endemic influenza with bronchitis in an infant seven months of age.

cases the pneumonia is of the pneumococcus variety. Especially severe are the cases of grippe which are ushered in with a chill, high fever, and cerebral symptoms, such as sopor, delirium, and rigidity of the neck muscles. In many of these cases examination of the chest reveals pneumonia. These cases are not so common among infants as among older children. Cases in which there is a cerebrospinal in-



fection in no way differ from cases of cerebrospinal meningitis due to the meningococcus or the pneumococcus. The endemic grippal forms of cerebrospinal meningitis may be caused by the influenza bacillus (Sänger) or the pneumococcus. The child at first complains of fatigue, and has a tendency to sleepiness, cries out and starts in its sleep, and suffers from intense headache. After a time vomiting

FIG. 43.



Endemic influenza, lobar pneumonia of the lower lobe of the right lung. Child two and one-half years of age.

with rigidity of the muscles of the neck sets in. These symptoms increase in intensity, sopor finally setting in with all the symptoms of a cerebrospinal meningitis. These cerebral cases are rare. A common form of grippal attack is that in which all the symptoms of nasopharyngeal inflammation are present. There is also mild bronchitis of the larger tubes. The temperature may fall to the

normal in the morning or toward noon, but toward evening it rises from one-half a degree to three degrees above the normal (Fig. 43). The child plays in the afebrile intervals. It may awake from sleep in a peevish, irritable mood, or may start in its sleep. These symptoms may continue for a week or longer. In many of these cases there is serous or purulent otitis media, or there may even be a mastoid inflammation from the outset. In other cases the patient has an intermittent or remittent fever. The fever, if a continued one, has morning or evening remissions. Examination of the heart may reveal an acute endocarditis, although marked symptoms of cardiac involvement may be absent.

Symptoms referable to the kidney have received little attention in text-books. In endemic grippe there is almost always a slight trace of albumin in the urine, which, as a rule, disappears at convalescence. Occasionally, there is a true nephritis, with casts, decreased secretion, and blood. Such cases have been described by Freeman. Of grave import are the cases of nephritis in endemic grippe which at first show a trace of albumin and a few hyaline, epithelial, and blood-casts, with a very small (microscopic) amount of blood in the urine. The urine is normal in amount. The condition is revealed only by the microscope. Edema is absent. The child is at first pale, but this pallor disappears later. The trace of albumin in the urine, however, with a few casts and blood-cells, persists for months. These cases have been described as "cyclic" albuminuria. They are really nephritis of an insidious character following endemic grippe.

I have seen cases of endemic grippe complicated with swelling of the parotid and submaxillary glands and of the lymph-nodes of the neck.

The **duration** of endemic grippe is from two or three days to as many weeks. I have seen cases present a temperature-curve for three weeks, but have not met the cases of protracted duration, with or without fever, described by Filatow, and would regard such cases as peculiar to the country of that author.

The **prognosis** of endemic grippe is favorable. If complications supervene, it varies with their nature.

The **diagnosis** presents no difficulties. In some cases the nervous symptoms may cause the physician to suspect meningitis when pneumonia is present. A careful physical examination will dispel the doubt. Meningitis and pneumonia may be present in the same case. Otitis may supervene without the presence of marked symptoms referable to the ear. An aural examination by an expert should be made in all cases in which fever persists and physical examination of the lungs and other organs fails to reveal abnormal conditions.

The **treatment** of la grippe is simple. At the outset in the



milder cases small doses of quinine are administered, to control the headache, restlessness, and fever. For the angina small doses of ferric chloride are given to infants every one to three hours. In older children, the throat is, in addition, sprayed two or three times daily with salt solution or a solution of boric acid or listerine. The fever is treated by sponging; packing or baths are rarely necessary. The bowels of infants are washed out with high enemata if diarrhoea sets in, and milk food is temporarily suspended. Pneumonia, if present, is treated as outlined in the section on that disease. Otitis should be treated by early incision of the drum-membrane, as even cases in which no pus, but only serum, is present are relieved by this procedure. With older children the use of phenacetin alone or in combination with monobromate of camphor is permissible if the headache and pains in the limbs are very troublesome. A grain of each may be given once or twice daily for a short time. The prostration is best combated by the use of strychnine alone or combined with caffeine. Whiskey is not well borne in these cases, since it is likely to cause gastro-intestinal symptoms.

### GLANDULAR FEVER.

(Pfeiffer.)

Glandular fever is a form of infection which manifests itself by an enlargement of the lymph-nodes of the neck, with accompanying enlargement of the liver and spleen, and an initial period of fever. It occurs from the second to the eighth year of life. During an extensive epidemic J. P. West observed it in the nursing infant.

The **etiology** is obscure. This disease is a species of infection or toxæmia. In some cases (West) there has been diarrhoea, in others constipation, and in most cases a slight injection of the nasopharynx. It is possible that the infectious agent gains access to the lymph-channels through the gut or nasopharynx. This would account for the involvement of the mesenteric glands, as observed by Pfeiffer, and for the infection of the nodes of the neck through the thoracic duct.

**Symptoms.**—After slight malaise, or even without prodromata, children are attacked with fever, restlessness, headache, vomiting, and pains in the limbs. After a few hours of these premonitory symptoms, swelling of the cervical glands on one or both sides is noticed. These glandular swellings extend from beneath the body of the jaw along and beneath the upper third of the sternomastoid muscle. The lymph-nodes beneath the muscle are also affected. After one or two days these glands or nodes not only increase in size, but nodes at the back of the neck and in the supra-clavicular region are also affected. In the cases recorded by West

the axillary and inguinal lymph-nodes were also involved. The temperature at first ranges from 102° to 104° F. (38.8° to 40° C.), but in from twenty-four to forty-eight hours it may fall by crisis. There is a slight redness of the pharynx or the color of the mucous membrane may be normal. There is pain on deglutition, and there may be a slight cough, but no distinct pulmonary affection. In both Pfeiffer's and West's cases the liver and spleen were enlarged. In the cases of Starck, Rauchfuss, and Protussow these enlargements were not always present.

The **lymph-nodes** may enlarge to the size of a pigeon's egg. The redness of the pharynx is disproportionate to the enlargement of the nodes (Rauchfuss), so that it is hardly permissible to speak of an anginal lymphadenitis, as in scarlet fever. In both Starck's and West's cases there was enlargement of the nodes, which were not painful, but sensitive to pressure. The swelling of the carotid lymph-nodes began, as a rule, after a few hours, was in most cases first visible on the left side of the neck, and reached its height from the second to the fourth day. The glands on the opposite side of the neck then became affected. The swelling rarely continues unilateral. It is uniform, as thick as an index-finger (West), and is composed of several nodes. There is a stiffness of the neck and also a sensation of choking. Suppuration is absent. There is in all cases a tenderness of the abdomen about the umbilicus, which, in Pfeiffer's opinion, indicates an infection of the mesenteric nodes. West found the mesenteric nodes enlarged in 37 cases.

**Diagnosis.**—The disease is readily differentiated from mumps. In some epidemics the submaxillary glands were involved, but never the parotid. The appearance of the swelling of the lymph-nodes first on one side, and then on the other side of the neck is characteristic, and should be differentiated from the glandular swellings occurring with grippal affections or pneumonia. Heubner has reported cases in which there was a complicating nephritis.

**Duration.**—The fever disappears after a few hours or may last two or three days. It may recur later. The glandular swellings, however, increase or persist nine to twenty-seven days, the average duration being sixteen days (West, Rauchfuss).

**Treatment.**—As the affection has a tendency to spontaneous recovery, the treatment is purely symptomatic.

### CEREBROSPINAL MENINGITIS.

Cerebrospinal meningitis is an acute infectious disease, of which the characteristic lesion is an exudative inflammation of the pia mater of the brain and spinal cord. It occurs both epidemically and sporadically.



**Etiology.**—Cerebrospinal meningitis, both in its epidemic and sporadic forms, is due to infection by the *Diplococcus meningitidis* of Leichtenstern, Weichselbaum, and Jäger. This diplococcus has the general form of the gonococcus, is decolorized by the Gram stain, and is present in the body of the pus-cell (intracellularis). Another group of cases of cerebrospinal type is caused by the *Diplococcus pneumoniae*. These cases have been described by Netter, Foa, and Bordoni-Uffreduzzi. They occur epidemically, but generally in combination with lobar or bronchopneumonia and as a complication of otitis media. The form of affection discussed in this section is the sporadic and epidemic cerebrospinal meningitis caused by the intracellular diplococcus of Weichselbaum, Jäger, and Heubner. In epidemics of this disease it is unusual for several members of a family to be attacked. The number of cases in an epidemic may number several hundred. The disease has no marked tendency to spread. In large cities sporadic cases occur in localities widely separated.

**Occurrence.**—Cerebrospinal meningitis may occur at a very early age. Rotch reported a case in an infant six days old. The youngest case met by the author was in an infant ten weeks of age. Of 111 cases recorded by Councilman, 29 occurred in infants and children. Males are more frequently attacked than females. Epidemics and sporadic cases occur in the winter and early spring.

**Morbid Anatomy.**—In certain sporadic cases there are very marked symptoms, and yet post-mortem examination will show the gross appearances of the brain and pia to be normal. Under the microscope, however, a slight infiltration with pus and fibrin and a new growth of cells resembling those of the pia are seen. In other cases there is an extensive infiltration of the pia with serum, fibrin, and pus. The exudation is especially profuse at the base of the brain and at the posterior surface of the cord (posterior basic meningitis). The ventricles are markedly distended with serum and pus (Delafield).

Among the associated lesions found are subserous punctate hemorrhages of the endocardium; petechiae in the skin; hyaline and granular degeneration of muscle; multiple abscesses on the surface of the body; suppuration of the joints; parenchymatous degeneration of the heart, liver, and kidneys; and swelling of the lymph-nodes and spleen. In all cases the *Diplococcus intracellularis* is found in the exudate on the pia and surface of the brain and in the ventricular fluid.

The **symptoms** in the epidemic and sporadic forms of the disease are similar.

Clinically, the cases which I have observed are divided into three distinct types: *a.* Those in which there is headache, with rigidity of the neck, fever, and delirium at night, but in which there are intervals during which the patients are rational, sit up in bed and

play with their toys. *b.* The foudroyant cases, in which death supervenes in a short time, the onset with early coma and rigidity of the neck and opisthotonos being characteristic. *c.* The common form, beginning with vomiting, headache, and delirium, accompanied by rigidity of the neck and stupor. In cases of the last type the patients can be roused at intervals.

The symptoms of invasion are as follows: An infant eight months of age develops a conjunctivitis (intracellular diplococcus). After a week marked cerebral symptoms—vomiting, convulsions, coma, rigidity of the muscles of the neck—appear, with a high febrile movement. In other cases there may be a history of a fall. Four or five days after the fall (the patient meanwhile attending school) headache, vomiting, and difficulty in deglutition set in, and after a day or two of these symptoms convulsions manifest themselves, with fever and rigidity of the neck. In other cases the onset of the disease is signalized by chills, fever, and vomiting, and there is occipital and frontal headache. The chills and fever are repeated daily, as in intermittent malarial fever. The neck becomes painful and rigid, and the patient is dull and apathetic. To these symptoms are added delirium and opisthotonos. (Plate IX.). Although the modes of invasion differ in certain respects, they resemble each other in a general way. In typical cases the symptoms are grouped as follows:

**Cerebral Symptoms.**—If the fontanelle is not closed, there will be tenseness, followed by bulging, even in the early stages, certainly by the fifth day. The patients usually have delirium and coma. In the milder cases headache is the principal symptom, and periods of consciousness alternate with those of stupor. There is hyperæsthesia of the surface, and the patients cry out if the bed is jarred or the skin touched. In some cases there are chills and convulsions, the latter being unilateral. There may be facial paralyses and hemiplegia in the later stages of the disease.

**Reflexes.**—The patellar reflex may be absent or exaggerated. Babinski's reflex is absent in most cases. Kernig's symptom is quite constant.

The **skin** may be the seat of successive attacks of erythema. Herpes labialis may be present. Tache cerebrale is always present.

The **joints** may be tender or swollen. I have never seen suppuration of the joints.

There may be **intramuscular abscess**.

**Eye Symptoms.**—I have seen an initial conjunctivitis. In Councilman's cases there were keratitis, strabismus, contraction, dilatation, and inequality of the pupils, neuritis of varying grades of the disc, atrophy, and purulent choroiditis. There is no appreciable impairment of vision in some cases. In a four months' baby, paralysis of the orbital muscles of one side appeared early.



PLATE IX.



Cerebro-spinal Meningitis (Meningococcus). Rigidity of the neck, opisthotonus, characteristic position of the arms. Infant eight months of age, fatal issue.

•

•

•

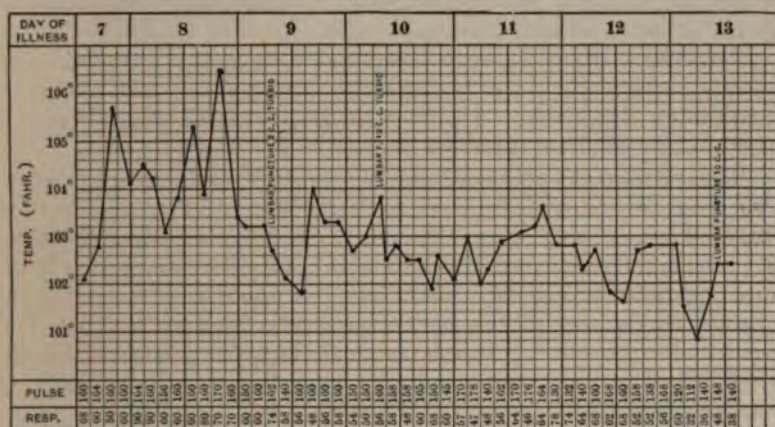
•



The nasal secretions in some cases show the intracellular diplococcus.

The temperature in cerebrospinal meningitis is not characteristic. There is generally a daily rise, which at the outset and in the subacute

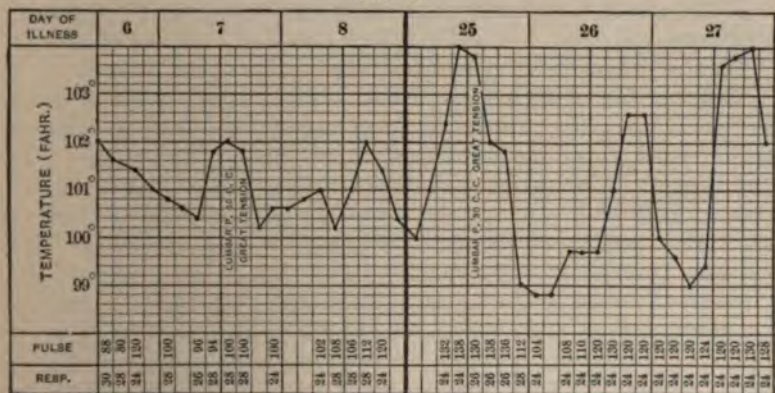
FIG. 44.



Cerebrospinal meningitis. Female infant, eight months of age. Unconscious on admission; fatal issue. (Meningococcus.)

stages of the disease may reach 106.4° F. (41.3° C.). In some patients the curve has a distinctly intermittent type, the temperature being normal or nearly so at some time in the twenty-four hours. In

FIG. 45.



Cerebrospinal meningitis. Female child, eight years of age. Temperature at two extremes of the illness. Recovery. (Meningococcus.)

other cases the temperature is persistently high, with remissions of a degree or more. On the other hand, an infant nine months of age was affected with cerebrospinal meningitis of the meningococcus

type, and during a course of two weeks showed no rise of temperature above the normal. This is, of course, exceptional. Chills or rigors are likely to recur in the course of the disease, and are followed by sharp rises in temperature.

**Circulation.**—The pulse is rapid and irregular, and the respiration as well as the pulse may be increased. Endocarditis, as indicated by the presence of murmurs, may exist.

**Paralyses.**—Hemiplegia or facial palsy, partial or complete, may occur, or there may only be weakness of the upper and lower extremities on either side. In the later stages of the disease there is incontinence of urine and feces.

The **respirations** are shallow but increased in frequency, and are also irregular, as will be seen by reference to the accompanying charts (Figs. 44 and 45). In some cases Cheyne-Stokes breathing is present, owing to cerebral pressure. In other cases this is not once apparent during the whole course of the disease. As the end approaches, the respirations may cease before the heart ceases to beat (ventricular pressure). In the terminal stage the respirations sometimes fall to 10 a minute and the pulse to 50 with the onset of general paralysis.

In very young infants not only do the fontanelles bulge, but the sutures are forced apart, as in hydrocephalus. In cases of this kind, on tapping the skull a percussion-wave is felt. Macewen's sign—a tympanitic percussion-note over the parietal or frontal bones (see page 260)—is also present in cases in which the ventricle is distended with fluid.

The **spleen** may be enlarged.

The **ear** may be the seat of otitis and mastoiditis. Deafness may result, and the child may walk with a swaying, tottering motion.

**Blood.**—There is leucocytosis at the height of the disease.

**Course and Prognosis.**—After symptoms are fully developed in typical cases, the patient lies unconscious, the head is retracted, and in some cases the back arched. Delirium is constant. The neck is rigid. The patient cries out with pain when moved, or even when undisturbed. During the rigors the patient becomes cyanosed, the heart is feeble, and the respiration shallow and irregular. There is always progressive emaciation. In some cases an abortive course is described: after headache, fever, and vomiting, convalescence sets in and the patient rapidly recovers. Other cases result fatally in a few days. Some run a course of from eight to twelve weeks, and finally recover. There are few recoveries of children under two years of age. According to Hirsch, the average mortality in older children is 40 per cent.

Recovery takes place without complications in some cases, but in others various cerebral lesions, such as idiocy, external hydrocephalus, blindness, and palsies, are manifest.





PLATE X.



Posterior Basic Meningitis. Child three years of age.



**Diagnosis.**—Cerebrospinal meningitis must be differentiated from tuberculous meningitis, typhoid fever, and pneumonia with cerebral symptoms.

It can be distinguished from tuberculous meningitis by its sudden onset, the presence of the high febrile movement, the early onset of rigidity of the neck and opisthotonos, and finally by examination of the fluid obtained by lumbar puncture.

**Lumbar Puncture.**—In cases of cerebrospinal meningitis this should be practised at first as a diagnostic, and subsequently as a therapeutic measure. By culture and cover-glass stain the characteristic intracellular diplococcus will be detected in the fluid withdrawn. I have found it present at the fifth and up to the thirty-seventh day of the disease.

In typhoid fever there are the characteristic Widal reaction, leucopænia instead of leucocytosis, and the enlarged spleen. The latter sign is of confirmatory value only.

In pneumonia examination of the lungs will reveal lesions. Cerebrospinal meningitis may coexist with pneumonia.

### Posterior Basic Meningitis.

(Barlow, Lees, Still.)

Posterior basic meningitis is now by English authors considered a form of cerebrospinal meningitis in which the exudate is most profuse at the base of the brain. Retraction of the head, marked opisthotonos, and emaciation are present early. These cases run a protracted course, and the symptoms are those of protracted cerebrospinal meningitis (Plate X.).

There are other forms of meningitis, due to the pyogenic bacteria, the staphylococcus or Streptococcus pyogenes. They are not primary forms of meningitis, but are secondary to otitis, pneumonia, traumatism, and the infectious diseases, such as scarlet fever and measles. If pneumonia exists as a complication, the symptoms are masked by those of this disease. The cerebral symptoms in otitis and pneumonia may lead to a diagnosis of meningitis. Caution should be exercised in making a diagnosis. I have seen cases in which rigidity of the head, hyperæsthesia, and delirium were present, but disappeared rapidly as soon as the otitis was properly treated or the pneumonia resolved. If meningitis is confined to the convexity, the symptoms in children are similar to those of the adult. If, however, a streptococcus, staphylococcus, or pneumococcus meningitis affects the base of the brain, the symptoms will resemble or be identical with those of the cerebrospinal type. Lumbar puncture should be performed in all cases of traumatic origin, in order to determine the nature of the process present. These forms of secondary meningitis are frequently fatal.

The **treatment** of cerebrospinal meningitis is symptomatic. The hair is cut short and an ice-bag placed on the head to relieve the headache and quiet delirium. When exudate is present, the patient is put on liberal doses of potassium iodide. Infants under a year are given grain j or ij (0.06 or 0.12) three or four times daily. The delirium is quieted with potassium bromide combined with chloral hydrate. The convulsions, as a rule, are not severe, and require only ordinary dosage of the bromides and chloral. The bowels are kept open by the administration of calomel every few days. The fever, as a rule, is not continuously high, and is treated with sponging. If rigors occur with depression of the heart, warm baths give great relief. The patients are put into a bath at 105° F. (40.5° C.) for five or ten minutes. The baths not only reduce the temperature, but also stimulate the heart. In extreme cardiac weakness small doses of camphor are indicated. In cases in which there are symptoms of cerebral pressure, such as extreme rigidity and opisthotonos, coma, delirium, bulging fontanelle, and chills and rises of temperature (suppuration), I perform lumbar puncture. By this procedure the cerebral pressure may be relieved for a short time, and the amount of purulent or bacterial exudate diminished. About 20 to 50 c.c. are withdrawn. In some cases the exudate is so thick and viscid that only a small quantity can be drawn off. The flow of fluid should never be aided by an aspirating syringe. In the experimental laboratory punctate hemorrhages in the medulla and cord have been caused in this way. Days should be allowed to elapse before the procedure is repeated, and it then should be repeated only when there is an exacerbation of symptoms. It should be done as early in the disease as possible, and under strict antiseptic precautions. When properly performed, no ill results follow.

### MUMPS.

(*Epidemic Parotitis.*)

Mumps is an infectious and contagious disease of the parotid gland, at times involving the other salivary glands as well as the testis or ovary.

**Etiology.**—Parotitis is endemic in large cities, and frequently becomes epidemic in schools and institutions where large numbers of children are congregated. It is most common among children of school age, because they are more exposed to infection than children at an earlier or later period of life. Girls and boys are attacked with the same frequency. It may occur in the newly born infant. The author has seen a case in an infant three weeks of age.

The essential cause of mumps is unknown. Laveran and Catlin describe micrococci which they found in the blood and in the glandu-



lar lymph of the parotid and testis. These micrococci were arranged in twos and fours, did not stain by the Gram method, and were 1 to 1.5 micromillimetres in diameter. Michaelis and Bein isolated an intracellular chain-forming diplococcus from Steno's duct. The theory thus far advanced is that these micro-organisms gain access to the parotid through the duct. The period of incubation, according to Rilliet and Lombard, may vary from seven to twenty-six days.

**Morbid Anatomy.**—As the disease is rarely if ever fatal, opportunities to determine the morbid conditions have been few. Virchow first described the condition of the gland as one of inflammatory serous and cellular infiltration of the intra-acinous and peri-acinous connective tissue. The outcome is resolution; induration rarely remains.

**Symptoms.**—There is a prodromal period, during which the patient is attacked with chilly sensations or a chill, and sometimes

FIG. 46.



Parotitis, bilateral. Boy, six years of age.

with vomiting. There is pain in the region of the ear, and also a ringing in the ears and deafness. There is also a febrile movement, the temperature in some cases mounting to 104° F. (40° C.). The temperature may be normal throughout the disease. There may be headache and loss of appetite. After these symptoms have lasted awhile, the face becomes swollen, as a rule on one side only (Fig. 46).

This swelling gives the face an uneven contour, and is the characteristic symptom. In older children it causes a feeling of tenseness and pain on mastication. Sometimes patients are averse to opening the mouth on account of the pain. In young infants there is drooling. In the majority of cases, after the swelling has lasted three or four days and is subsiding, the opposite side becomes affected. In addition to the swelling of the parotid there is also intumescence of the lymph-nodes of the neck at the angle of the jaw and of the node on the parotid gland in front of the ear. Frequently the submaxillary glands are also swollen, giving the whole face a rounded

FIG. 47.



Parotitis involving the submaxillary glands, lateral view. Boy, four years of age.

contour. In most cases the general condition of the patients is good and there is very little discomfort. Other cases have considerable pain and constitutional disturbance. In all my cases there was distinct angina and swelling of the tonsils. In a newly born baby there was swelling of the tissues underneath the jaw and about the larynx, with croupy breathing indicating œdema of the mucous membrane of the larynx.

English authors have described cases in which the submaxillary glands alone were involved, the inflammation being strictly limited to the glands on both sides (Fig. 47). I have seen a few cases of this kind.



**Complications.**—The testes and epididymis in boys and the ovaries and glands of Bartholini in girls may become affected. There may be ardor urinæ and a urethral discharge. These complications are not so common as the text-books declare. Hydrocele may occur with the orchitis. I have seen a case of this kind in a very young infant. The urine may show a trace of albumin, or in very rare cases there may be blood in the urine. Endocarditis, pericarditis, rheumatism, and osteomyelitis have been reported as complications, but the author has never met such cases. Parotitis complicating pneumonia has been observed in a boy of six years, and in another case otitis and parotitis were present at the same time. In rare cases the breasts and lachrymal glands are affected. Parotitis may be a complication of typhoid fever, measles, varicella, and influenza.

**Course.**—The disease is at its height in from three to six days, and runs its course in from seven to fourteen days. Mild cases may

FIG. 48.



Angiotoma of the parotid simulating mumps.

last only two days. Severe cases are rare. These present cerebral symptoms, and swelling of the tissues about the neck simulating angina Ludovici, with considerable dyspnœa. Cases of recurrent mumps, continuing for from four to six weeks, are recorded. When suppuration occurs, it is probably the result of some mixed infection.

The **diagnosis** is not difficult. Uncertainty as to whether the parotid is affected or not will be dispelled by drawing a line parallel with the lower border of the jaw; the parotid swelling will be above the line and the lymph-nodes of the neck below it (Fig. 46). In swelling of the mastoid region the ear is raised from the skull, while in parotid swelling, even if it occur behind the ear, that organ remains in its normal position. The swelling of parotitis never fluctuates, but is elastic in character.

The **prognosis** of mumps is good; the majority of cases recover without complications. If the kidneys and endocardium and pericardium are affected, the prognosis will be influenced by the course of these affections. I have never known epidemic parotitis to result fatally.

**Treatment.**—The patients are isolated and kept in bed as long as symptoms are present. The parotid is anointed twice daily with warm oil of hyoscyamus and covered with cotton. The bowels should be regulated with a saline cathartic. The diet should be assimilable. The affection cannot be controlled by means of drugs. Pain and fever are treated on general principles.

### PERTUSSIS CONVULSIVA.

(*Whooping-cough.*)

Pertussis is an acute specific infectious disease, caused by a micro-organism, probably of the influenza group. It is characterized in the majority of cases by a spasmodic cough accompanied by a so-called whoop.

Pertussis is not only infectious, but it is also contagious. It is propagated through the atmosphere in schools and public places, the air of which is contaminated with the specific agent of the disease. The micro-organism is thought to exist in the sputum and the secretions of the nasal and air-passages of the patient. The disease is especially contagious at the height of the attack. There is reason to believe that the cough of the first or catarrhal stage is highly contagious. The sputum in the stage of decline is also, in my opinion, capable of conveying the disease to others, since it contains the specific micro-organism.

**Occurrence.**—Pertussis prevails in all countries and climates. It is most frequent during the winter and spring months. It is always endemic in large cities, but, like scarlet fever, becomes at times so prevalent as to be epidemic. Pertussis is essentially a disease of infancy and childhood, but the individual is not exempt at any age. It has been seen in the newly born infant. I have found the disease slightly more frequent in females than in males (1009 out of 1820 cases). Twenty-two cases occurred in infants between one and two months of age. The majority of cases (1343)



occurred between the sixth month and the fifth year. The disease is most frequent between the first and the second year (404); next most frequent between the sixth and twelfth month. After the fifth year the frequency diminishes up to the tenth year, after which the disease is very infrequent. Not every one who is exposed contracts the disease. One attack does not necessarily confer immunity, but cases of second attack are rare. It has been observed that pertussis, measles, and influenza frequently follow one another in epidemic form.

**Etiology and Bacteriology.**—The essential cause of pertussis was believed by Deichler and Kurloff to be a protozoa-like body which they found in the sputum. Afanassjew and Szemetzchenko isolated a bacillus from the sputum. It occurred singly, in pairs or chains, and measured 0.6 to 2.2 micromillimetres in length. The more recent researches on the bacteriology of pertussis are those of Czapelewski, Hensel, and Koplik. Czapelewski and Hensel described in 1897 a non-motile "pole bacterium" or bacillus resembling the influenza bacillus. I at the same time described in the sputum a finely punctate, thin, minute bacillus, 0.8 to 1.7 micromillimetres in length, resembling the influenza bacillus, and staining like that or like the diphtheria bacillus. This bacillus was found recently by Luzatto in cases occurring in an epidemic of pertussis in the city of Graz. It is classified by him as belonging to the influenza group. Positive proof that this bacillus is the cause of pertussis is lacking, since the disease has not as yet been produced experimentally. Evidence simply points toward a bacillus of the influenza group constantly found in the sputum.

**Morbid Anatomy.**—Post-mortem examination reveals marked inflammation of the nasal passages, bronchopneumonia, and empyema or simple fibrinous or serous pleurisy. Emphysema as a result of rupture of the lung-tissues has been reported by Northrup, who describes the lungs of an infant seven months old as being studded with cavities measuring one-half a centimetre to two centimetres in diameter. The lungs looked like parchment filled with bubbles. Hemorrhages in the eye, ear, and brain are a feature of the morbid anatomy of fatal cases.

**Symptoms.**—There is undoubtedly a period of incubation, but its length is undetermined, and it can only be said that, if the disease is due to the invasion of a micro-organism, some time must elapse between the invasion and appearance of symptoms. After the appearance of the symptoms there are three stages—the catarrhal, the spasmodic, and the stage of decline. There is no sharp line of demarcation between these stages.

**Catarrhal Stage.**—This stage in some children is characterized by a cough which is especially troublesome at night, and has sometimes a croupy character. The peculiar nature of the cough becomes ap-

parent when after a few days it becomes more troublesome instead of subsiding. After four or five days it may be accompanied by vomiting once or twice a day, especially if the paroxysm occurs after meals. Examination of the chest may fail to reveal bronchitis. This negative sign is of great value. As the case passes into the spasmodic stage it is noticed that the paroxysms of coughing last longer, and that the child becomes red in the face and expectorates a larger amount of mucus than in ordinary catarrhal conditions. This period of cough without a whoop may last five to twelve days. I have seen many cases in which the whoop was absent in the whole course of the affection. The child had what might be regarded as a severe spasmodic cough followed by vomiting. Fever is present as a rule only during the first few days. It may be remittent and slight. If bronchitis complicates this stage of the disease, there may be a daily rise of one or more degrees in temperature. Usually toward the close of the catarrhal stage the incessant cough causes slight puffiness of the eyelids and slight œdema of the tissues of the face.

The **spasmodic stage** is distinguished by the presence of the characteristic whoop. The cough becomes of a more pronounced spasmodic type. The child has distinct paroxysms, which begin with an inspiration, followed by several expulsive explosive coughs, after which there is a deep, long-drawn inspiration, which is characterized by a loud crowing called the whoop. After one paroxysm has ended, it may be followed by a number of similar ones. When a paroxysm is impending the face assumes an anxious expression, and the child runs to the nearest person or to some article of furniture and grasps it with both hands. The paroxysm is sometimes so severe that the child will fall prostrate or claw the air convulsively. In the severest and most dangerous type a convulsion supervenes. In moderately severe types of the disease the child's face is red or livid, the eyes bulge, and at the end of the paroxysm a quantity of tenacious mucoid or mucopurulent sputum is expectorated. In other cases there is vomiting at the end of the paroxysm. In the intervals the face is livid or pale, or the eyelids are puffy and the face œdematous. In some cases there are punctate hemorrhages on the face, especially about the eyes and temples. There may be chemosis of the conjunctivæ as a result of the bursting of bloodvessels. At this period there is in the majority of cases an accompanying bronchitis, with slight rise of temperature during the day. At first the paroxysms occurring during the twenty-four hours may be few; in some cases they never become frequent, but as a rule they increase in number, so that the patient may have from twenty to one hundred in the twenty-four hours. This stage gradually declines, the number of paroxysms diminishing daily in number and severity. They may subside suddenly or gradually after



from four to twelve weeks. The whoop may at times reappear. After the disappearance of the whoop a cough persists for days or even weeks, or it may entirely disappear and suddenly recur with the whoop. It is characteristic of the spasmodic period of the disease that the paroxysms should be more harassing at night than during the day.

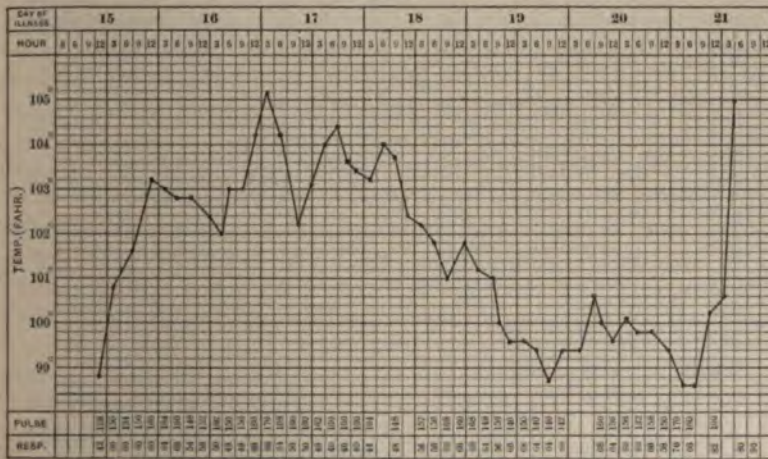
**Other Symptoms.**—In all cases of pertussis, even in the absence of complications, there is a slight increase in the number of respirations. In cases of even moderate severity the heart impulse is weak, and in exceptional cases the area of superficial cardiac dullness is larger than normal, indicating dilatation of a moderate degree. The pulse is irregular in force and rhythm, and is distinctly more dirotic than normal. In other words, there is a condition of heart-strain, which is evinced by dyspnoea (even in the absence of exertion), oedema of the face, and cyanosis.

**Kidneys.**—In the majority of cases a trace of albumin is present in the urine; in others, a few hyaline casts. Blood in the urine is seen in rare cases.

**Blood.**—Leucocytosis of the polynuclear type is usually present in the second week of the disease.

**Complications.**—One of the most common complications of pertussis is bronchitis. It may be mild or severe. In the severer

FIG. 49.



Pertussis; disseminated bronchopneumonia in both lungs. Infant eight months of age. Fatal termination.

form the smaller bronchi are affected, with accompanying bronchopneumonia (Fig. 49). The physical signs are the same as in simple bronchitis and pneumonia without pertussis. In some cases the bronchopneumonia pursues a subacute or persistent course. If reso-

lution takes place, other areas become consolidated. Emaciation is sometimes extreme. Emphysema is frequently present. Bursting of the air-vesicles may cause pneumothorax, or air may escape into the mediastinum and thence into the neck and into the subcutaneous tissue of the whole trunk.

**Hemorrhages.**—During a paroxysm there may be epistaxis, conjunctival hemorrhage, bleeding from the ears, and petechiæ on the face and body.

**Nervous System.**—**CONVULSIONS**, either general or localized, may complicate pertussis. In the former case the outlook is grave, death usually taking place within twenty-four to forty-eight hours.

**PSYCHOSES**, such as melancholia and hallucinations, may complicate pertussis. Monoplegia, hemiplegia, or paraplegia, localized facial and oculomotor paralyses, sudden total blindness, deafness, cerebral hemorrhages, hemianæsthesia, and aphasia have been observed.

**Gastro-enteritis** of a fatal type may ensue.

An attack of pertussis may favor the invasion of the tubercle bacillus. This may have been previously present in the bronchial lymph-nodes or elsewhere in the body, or it may be received into the body during the attack or afterward. In such cases tuberculosis of the lung or other organs, such as the peritoneum, develops.

**Diagnosis.**—If a cough fails to improve and is especially harassing at night, later in the disease becoming paroxysmal, if the face becomes livid during the paroxysm, if the patient vomits after coughing, pertussis should be suspected and precautions taken to prevent its spread. As a rule, examination of the chest is negative in the first stage. The absence of bronchitis and the presence of a cough of the character described, are characteristic of pertussis. The presence of the whoop dispels all doubt.

Infants who have the incisor teeth and other children may, after the pertussis has lasted for a week, develop an ulceration of the frenum of the tongue, which is called a dentition ulcer. It is caused by friction of the frænum linguæ with the edges of the teeth during the act of coughing. These ulcerations are not diagnostic of the disease; many cases do not show them, and on the other hand they frequently occur in coughs of other forms.

**Mortality and Prognosis.**—The mortality of pertussis is greatest during the first year of life (25 per cent., Voit). Between the first and the fifth year it is about 5 per cent., and from this time to the tenth year, 1 per cent. (Monti). The occurrence of pneumonia in children under two years of age adds largely to the mortality. Rachitis or marasmus will militate against recovery. Hygienic surroundings render the prognosis more favorable.

**Treatment.**—**Prophylaxis.**—The patient should be isolated, and should sleep in a large, well-ventilated room. During the day the sleeping-room may be filled for an hour with the vapor of formalin



(set free by means of a small formalin lamp). The object is to destroy suspended germs. If two communicating rooms are available, they may be occupied alternately every twenty-four hours, the unoccupied room being fully ventilated in the interval. In this way reinfection may be avoided.

In spring and summer, if the weather is favorable, the children should be constantly in the open air during the day. In large cities the mother is directed to take the child into the park. When in the open air the paroxysms are usually notably lessened. The child should be warmly clad in winter. Sea air seems to aggravate some cases and benefit others. Pine woods and moderately high altitudes are probably the most beneficial, for the patients are not exposed to the unfavorable climatic conditions peculiar to the seacoast.

**Medicinal treatment** consists of inhalations, topical applications, and internal remedies. Simply to enumerate all the remedies which have been proposed and used in pertussis, would take up the space of a monograph. Inhalation of ozone has been advocated by Caillé. The remedy is expensive and the apparatus not readily procurable. Inhalation of a mixture of 20 per cent. nitrous oxide and 80 per cent. oxygen is beneficial in cases in which the heart is weak. The inhalations are given with a cone for ten minutes twice daily. Insufflation of quinine or other drugs has not proved beneficial. The practice seemed to intensify the paroxysms. Prior, Coggeshall, and others have proposed the application of solutions of cocaine, 4 per cent. to 10 per cent., to the nares and throat. I have had no experience with this method, nor with the local application of antitussin.

If the cough is very troublesome, I first endeavor to control it with full doses of antipyrin combined with tincture of digitalis. The digitalis, in doses of a drop or two several times daily, supports the heart, as is shown by the rapid disappearance of the œdema and cyanosis after its administration. Antipyrin is given in doses of grain  $j$  (0.06) for every year of age up to grains  $v$  (0.3) every three hours. If the cough is not perceptibly relieved by this remedy after forty-eight hours, I suspend its use, and give codeine in full doses every three hours. Codeine is to be preferred to morphine, which is advocated by Henoch. If vomiting is severe, the food is given in very small quantities in fluid form every few hours. By this method food is retained and absorbed, whereas a full meal is invariably rejected. The use of belladonna has not impressed me favorably. In several cases it seemed to aggravate the cough by drying the laryngeal mucous membrane. Bromoform I consider dangerous and of questionable utility. Quinine in full doses three or four times daily is a favorite remedy with many pediatricists. Vaccination and the injection of diphtheria serum have been proposed to abort the disease. I have had no experience with the serum treatment. In a

word, the treatment of pertussis consists in applying the rules of hygiene, in mitigating the cough with antipyrin or preferably codeine, and in supporting the heart with digitalis. The complications should be treated on the principles laid down in the sections on Bronchitis, Pneumonia, and Pleurisy.

### DIPHTHERIA.

Diphtheria is a contagious febrile disease which affects the throat and air-passages. It is characterized by the formation of a pseudomembrane on the parts affected. The disease manifests itself by a local lesion and general symptoms caused by the entrance of toxins and, at times, of bacteria into the blood and lymph.

**Age and Occurrence.**—Although diphtheria is uncommon in the newly born infant, statistics of large numbers of cases show a certain percentage in these subjects; thus, of 547 cases reported by Monti, the newly born number 24, and in Baginsky's statistics several cases are noted. The disease is more frequent from the first to the third month than from the third to the tenth month (Monti). The largest number of cases occur from the second to the sixth year (40 to 63 per cent.) (Monti, Baginsky).

According to Seitz, it is slightly more frequent among boys than girls. Strong as well as weakly children are attacked. Children who suffer from nervous affections, such as poliomyelitis, are more likely to contract the disease than others (Baginsky). All exposed to infection do not contract the disease, because some individuals are immune. Escherich and Fischl have proved that the blood of convalescents contains antitoxic elements. Cases of several attacks in the same individual are not uncommon. Racial peculiarities have no influence.

Diphtheria is prevalent in all parts of the world and epidemics occur at all seasons of the year. It is more common among the poorer classes, not on account of uncleanness, but as a result of overcrowding.

**Contagion.**—Diphtheria is contagious from person to person, and may be conveyed by any one who has been in the room occupied by a patient with the disease. Mild cases may give rise to fatal cases. The disease is infectious, spreading through families and schools, and may be conveyed through the medium of toys, clothes, and in milk.

**Etiology.**—The essential cause of diphtheria is a bacillus, the *Bacillus diphtheriae*, which was first noted in stained specimens by Klebs in 1882. Löffler first isolated and accurately described it in 1884. It is present in all cases of true diphtheria of Bretonneau. In the 3 per cent. of cases in which it is reported absent there is good reason to believe that failure to establish its



presence was due to imperfect technique. The bacillus is non-motile, twice as thick and about as long as the tubercle bacillus, thickened at the extremities, has no spores, and in some forms has been described as branching. It is very resistant, adheres to clothes and candy, and has been found in milk. It will retain vitality a long time in dried membrane (seventeen weeks), as has been shown by Roux and Yersin. It has been detected nine weeks after the disappearance of the membrane from the throat. It is found present with other bacteria, principally staphylococci and streptococci, pneumococci, *Bacillus coli commune*, *pyocyaneus*, *proteus*, and *sprue*. It has been found by Roux and Yersin in the throats of perfectly healthy

FIG. 50.



FIG. 51.



The *Bacillus diphtheriae* (Klebs-Löffler).

FIG. 50.—Pure culture, photomicrograph.  
× 1000.

FIG. 51.—Pure culture, photomicrograph.  
× 1000. Shows the irregular beaded stain.

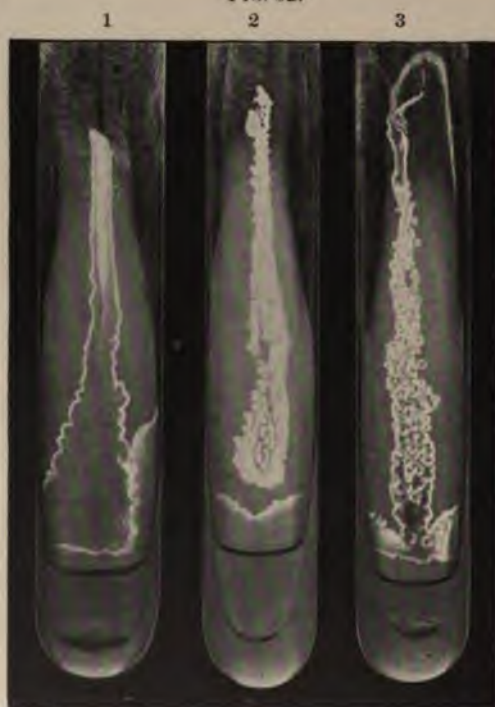
individuals, and may be present without the formation of a membrane. It has been shown that this bacillus forms toxins of very positive action. According to Sidney, the toxins of diphtheria may be divided into albuminoses and organic acids.

The pseudobacillus of diphtheria was first isolated by Hoffman. In its growth and staining properties it is identical with the true diphtheria bacillus, but is not virulent to animals. Roux and Yersin regard it as a weakened diphtheria bacillus. Others believe that it bears no relation to the true bacillus. It is found associated with the true bacillus, and also in cases of diphtheria after this bacillus has disappeared from the throat (Koplik). Some authors have

given the name pseudodiphtheria bacillus to another variety of bacilli, but this term should be strictly limited to the form described above.

**General Infection with the Bacillus Diphtheriæ alone and with Other Bacteria.** The bacillus of diphtheria was first demonstrated by Frosch (1895) in the heart's blood, liver, spleen, kidneys, and lymph-nodes. Since then, Kolisko, Paltauf, Schmorl, Booker, Councilman, Mallory, and Wright have demonstrated its presence in the blood and internal

FIG. 52.



1 and 3. Cultures of the pseudobacillus of diphtheria on agar, showing the diffuse character of the growth. 2. Growth of *Bacillus diphtheriæ* (Klebs-Löffler) on the same medium. It is a delicate growth in colonies.

organs in fatal cases of diphtheria. The work of Councilman and his pupils is the most recent and complete on this subject. They show that the bacillus may occur alone or in association with streptococci or staphylococci in the blood, lungs, liver, spleen, and kidney. It is more likely to be found alone in fatal cases of uncomplicated diphtheria. The mixed infections with streptococci and other bacteria occur in diseases, such as scarlet fever and measles, which may be complicated with diphtheria. The investigators just mentioned found endocarditis, bronchopneumonia, empyema, mastoid disease,



and thrombosis of the sinuses due to the diphtheria bacillus. The bacillus was found also in the pus of acute abscesses in various localities.

**Morbid Anatomy.**—In fatal cases the membrane appears as a thick brownish or grayish-brown mass. It is sometimes present as a thin whitish pellicle, and occasionally is almost black. It may be friable or as resistant as cartilage, and may extend over the tonsils, palate, pharynx, base of tongue, epiglottis, and trachea. The areas not covered by membrane are injected, and may be the seat of hemorrhages. The tonsils are enlarged and bluish red. In the gangrenous forms the tonsils, soft palate, and uvula may be converted into necrotic masses. The nasal passages may show membranous deposit. The epiglottis and vocal cords are thickened. The tracheal mucous membrane is hyperæmic and swollen, there may be adherent membrane, or the pseudomembrane may be loose and curled up in the lumen of the trachea.

The membrane itself has been described by Virchow as croupous and diphtheritic. Councilman is of the opinion that little is to be gained by adhering to the old classification of croupous and diphtheritic membranes. Baginsky also describes forms of diphtheria in which the membrane possessed both croupous and diphtheritic structural characteristics. According to Councilman, the first step in the formation of the membrane is a degeneration and necrosis of epithelium, preceded by a proliferation of the nuclei of the cells. Detritus and hyaline masses result. An inflammatory exudate rich in fibrin is thrown out from the underlying tissue. The fibrin forms in part a reticulum enclosing cells and degenerated epithelium, and in part a hyaline reticulated membrane. The hyaline membrane is formed on surfaces which are covered with several layers of epithelial cells. Fibrinous membrane is formed on the surface and in the tissue. By constant accretions thick masses are formed. The membrane is never formed on an intact epithelium, but may extend over it. There is nothing specific in the diphtheritic membrane. The connective tissue and the bloodvessels beneath the membrane may be the seat of hyaline degeneration. The mucous glands are degenerated.

The diphtheria bacilli are found growing in the necrotic tissue and in the exudation, never in the living tissue or in epithelium undergoing primary degenerative changes. In exceptional cases they may be found enclosed in pus-cells and necrotic epithelium. They are found in masses, and when deeply situated have been covered up by later formation of membrane.

**Heart.**—Councilman, Mallory, and Pearce have recently described the myocarditis sometimes complicating diphtheria. There is a fatty change in foci or in more diffuse areas in the muscle-fibre. In another form of myocarditis there are interstitial changes, consisting of focal collections of plasma and lymphoid cells, and the forma-

tion of new connective tissue, resulting in some cases in a fibrous myocarditis. These pathologic changes are due to the action of the diphtheria toxins on the heart-muscle.

**The Lungs.**—Councilman states that the most common lesion in fatal cases is a bronchopneumonia, lobar pneumonia never being present. The process begins in an infection of the atria. The bacteria found in the lung, and which are present independently of the character of the lesion, are the pneumococcus (rarely), *Streptococcus pyogenes*, and the diphtheria bacillus. Marrow-cells are found in the capillaries, and thrombi in the larger vessels. The lymphatics are dilated and contain fibrin and cells.

The **spleen** macroscopically is normal; microscopically, the lymph-nodules are more prominent than is normal, and contain foci of epithelioid cells. The vessels are the seat of hyaline degeneration, and in the later stages contain large numbers of plasma-cells. Some of the nodes may be the seat of necrosis and abscess.

**Liver.**—The changes in this viscus are due to the action of toxins, and consist of parenchymatous degeneration and necroses, seen especially in the centre of the lobules. There is slight hyaline degeneration of the capillaries.

**Kidneys.**—There may be simple degeneration or acute nephritis. The severe forms of nephritis are found in the cases which are quickly fatal (Councilman). The interstitial and glomerular changes are more common in older children and in protracted cases. There is no specific form of nephritis in diphtheria, and all the changes are due to the action of toxins.

**Lymph-nodes.**—The mesenteric lymph-nodes, the nodes at the angle of the jaw and in the retropharynx and œsophagus are enlarged, and may undergo necrotic changes (Flexner). Councilman, Mallory, and Pearce describe the changes in the lymph-nodes as being more marked in those nearest the lesion. There are congestion, hemorrhages, and diffuse and circumscribed necrosis. In addition there is a formation of foci resembling miliary tubercles, and composed of epithelioid cells which undergo degeneration, forming granular detritus. Bacteria are not found in the nodes. The changes are due to the toxins.

**Nerves.**—There are fibrillation, increase of the cells of the sheath of Schwann, fatty degeneration of the axis-cylinder, hemorrhages, and nodular degeneration of the nerve-sheaths. In the spine there are infiltration of the meninges, hemorrhages, and degeneration of the anterior horns. Degenerative oculomotor changes are present. There are dilatation and round-cell infiltration around the central canal of the cord.

**Stomach.**—Diphtheritic membrane in the stomach occurring in cases of diphtheria has been described by Smirnow and Councilman. Of 220 cases reported by the latter, 5 showed the pres-



observer. During the paroxysms of coughing membranous casts are expelled from the larynx. The membrane may extend downward, involving the trachea and bronchi, casts of which may be expelled. The lungs may become involved, and in severe cases are the seat of a bronchopneumonia of streptococcic nature. With this there may be compensatory emphysema. The urine may show the existence of slight or extensive nephritis, or may be normal in every respect.

Especially deceptive are those cases of membranous laryngeal diphtheria or croup whose onset closely resembles that of so-called catarrhal laryngitis. In these the symptoms may develop suddenly, and within twenty-four hours the patient presents all the symptoms of laryngeal obstruction (croup d'emblée of the French). Inspection may show little variation from the normal appearances in the pharynx. We should be cautious not to assume that no membrane is present in the larynx. Cases have been recorded in which laryngoscopic examination failed to show membrane in the larynx, but in which post mortem it was found present beneath the cords and in the trachea.

**Course and Duration.**—In the mildest and purely local forms the disease reaches its height in from two to four days; the temperature then drops to the normal and convalescence is established. In the severe septic forms the membrane spreads from the tonsils to the pharynx, and the disease attains its full development in from five to eight days. The temperature falls by lysis or crisis, and convalescence is established. If the case is very severe, the disease shows no tendency to limit itself, the toxæmia is extreme and the involvement of the lymph-nodes is very great. Death may ensue in from a week to fourteen days. In some very malignant cases death may ensue in from three to four days after the onset of the disease. The laryngeal diphtheritic croup reaches its full development as a rule early—within three days. The disease may then retrograde under treatment or may advance into the trachea and bronchi, and cause death in a variable length of time.

The **complications** include bronchopneumonia, pleuritis, gastro-enteritis, retropharyngeal abscess, suppuration or necrotic destruction of the lymph-nodes of the neck, nephritis, cardiac paralysis, early and late (or post-diphtheritic) general paralysis, and diphtheria of the eyes, skin, and vulva.

**Bronchopneumonia and Pleuritis.**—Bronchopneumonia is found in from 50 per cent. (Baginsky) to 80 per cent. (Talamon) of the autopsies on children who have died of diphtheria. It results from extension of the disease from the trachea into the smaller bronchi and alveoli of the lung, and is therefore always a true bronchopneumonia. Through the investigations of Löffler, Flexner, Northrup, and Prudden, it has been proved that the diphtheria bacillus, the *Streptococcus pyogenes*, the *Staphylococcus aureus*, and the pneumococcus are the exciting causes of it.

PLATE XI.



G Dupuy

1. Tonsillar Diphtheria, with a small patch of membrane on the uvula.
2. Tonsillar Diphtheria, with a patch of membrane on the pillars of the fauces.
3. Acute Follicular Amygdalitis, which may be diphtheritic.



ence of membrane to a greater or less extent. The membrane either covered the whole surface or formed patches or streaks over the rugæ. The mucous membrane was swollen, hyperæmic, or hemorrhagic.

**The Middle Ear.**—Of 144 cases reported by Councilman, Mallory, and Pearce, 86 showed involvement of the middle ear on one or both sides; in 7 the mastoid was affected. The inflammatory products were serum or pus. The organism most constantly present was the streptococcus, but the diphtheria bacillus has been found, as have also the staphylococcus and pneumococcus.

**The Blood.**—The specific gravity is increased at the height of the disease. In mild cases it is not perceptibly changed; in severe septic cases it may range from 1054 to 1060 (Baginsky). Hæmoglobin is reduced only in severe cases of protracted course. Leucocytosis is not marked in mild cases, but in severe septic forms an increase of the white blood-cells has been observed by Felsenthal and Monti. In malignant cases there is a reduction in the number of red blood-cells (Ewing, Billings, Morse).

**Symptoms.**—Clinically, it is convenient to divide diphtheria into the purely local forms with few constitutional symptoms, the local forms with symptoms of marked toxæmia or septic forms, and the laryngeal forms.

**Purely Local Forms with Slight Constitutional Disturbances.**—In diphtheria sine membrana, synanche contagiosa (Senator), or catarrhal diphtheria, there may be no formation of membrane, the fauces showing only an angina of varying severity. In some cases there is the picture of a follicular or lacunar amygdalitis. Macroscopically there is nothing to show that the process is diphtheritic (Plate XI.). In other forms the membrane is present on the tonsils as specks or strips of exudate, or white or greenish pultaceous masses which may extend to the uvula, or there may be spots or extensive plaques on the posterior pharyngeal wall. In other mild cases the process is confined to a small necrotic excavated area in one or the other tonsil, as described by Hænoch. In still other forms the membrane may cover both tonsils, and extend over the soft palate and pillars of the fauces. In these forms of localized diphtheria the nares are seldom involved.

In these localized forms of diphtheria the infant or child may present few symptoms pointing to the throat affection. Unless the physician be systematic in his methods of examination, he may fail to inspect the throat at his first visit, and the diphtheria may thus escape detection. The nursling in this as in the non-diphtheritic affection, may refuse to take the breast. The movements are greenish, and have an offensive odor, or may be diarrheal. There are fever and restlessness. Inspection will reveal slight or marked swelling of the lymph-nodes at the angle of the jaw. The temper-

the pneumonia resulting from the diphtheritic or pseudodiphtheritic processes complicating scarlet fever and measles, Prudden and Northrup have shown that the *Streptococcus pyogenes* is an active causal agent. The onset of a complicating pneumonia is generally indicated by an exacerbation of the dyspnœa, fever, and cough. The prostration is also more marked. Auscultation of the inferior lateral or posterior parts of the chest on one or both sides reveals the presence of bronchopneumonia; while resolution is taking place in one part of the lung, other areas are being involved. Thus an apparent improvement may be followed by a rapid rise of temperature, increased dyspnœa, and rapid pulse. This form of bronchopneumonia may be complicated by pleuritis of a serous, serofibrinous, purulent, or hemorrhagic type.

**Gastro-enteritis.**—In nurslings there is frequently a diarrhœa with green stools and vomiting. In some cases these symptoms may become severe. Extension of the membrane into the œsophagus, stomach, and gut may take place, with a fatal result. The cases of simple diarrhœa are directly due to the swallowing of bacteria from the mouth and fauces. The diarrhœa may be so severe as to become one of the leading features of the disease.

**Retropharyngeal abscess** occurs in the tonsillar and pharyngeal forms of diphtheria as a result of infection of the retropharyngeal lymph-nodes by streptococci.

**Nephritis** may be absent, slight, or severe. Baginsky found it present in 42 per cent. of his cases. In the majority of cases of even mild diphtheria there is albuminuria; in some the urine may, in addition, contain casts, blood-cells, renal epithelium, and leucocytes, showing grave lesions of the kidneys.

**Heart paralysis.** Of greatest clinical significance is the cardiac diphtheritic paralysis, which may become apparent either early during the course of the disease or later on in convalescence. The early form may set in while the membrane is still visible in the throat. It occurs in the septic forms of the disease. These are the severe cases. The children show great prostration and apathy; the pulse is rapid and irregular; the heart-sounds, especially the muscular sound, is indistinct; the pulse is feeble and flickering; there are vomiting and abdominal pain. These symptoms may repeat themselves in attacks, until finally the patient dies with all the symptoms of collapse, such as cool extremities and shallow respirations. In such cases there is, as a rule, a marked nephritis. In the late cases the symptoms of cardiac failure appear from the second week of the disease to the seventh week of the convalescence. *The membrane has disappeared from the throat. There may be no premonitory symptoms, or there may have been a slight blowing*



and the muscular sound is weak; the pulse becomes small and either slow or rapid (tachycardia). There may be attacks of syncope, during which the patients vomit, complain of abdominal pain, and refuse medicine and nourishment. Sudden cardiac failure and death without symptoms, premonitory or otherwise, may occur in the period of convalescence.

Mild forms of cardiac irregularity which do not eventually prove fatal are seen in the beginning of convalescence. The severe forms of cardiac paralysis set in with symptoms of the early cases. These symptoms may have been preceded by the milder symptoms of cardiac irregularity. There is slight albuminuria. Suddenly, while in apparent good health, the patients complain of dyspnoea and pain in the stomach. The lips become cyanosed and the extremities cool, the pulse thready, the heart impulse weak, the heart-sounds scarcely audible; the heart may be rapid or as slow as 40 to 50 beats per minute. Vomiting is repeated, and in some cases the liver is enlarged. The patients may survive one or two such attacks, only to succumb finally. In the early forms of cardiac paralysis there may be no gross lesions in the heart-muscle. In the later forms the lesions are more apparent. There are fatty parenchymatous changes. In other cases there may in addition be changes in the vagi.

**Diphtheritic Paralysis.**—Paralyses are the result of the action of the toxins of the *Bacillus diphtheriae* on the nerve-trunks and tissues of the general nervous system. The paralysis may occur in the course of the disease or during convalescence. When the paralysis occurs early, it affects the velum pendulum palati. In cases which result fatally the heart becomes affected, pneumonia caused by the passage of food into the larynx develops, or the paralysis may become general. In the latter case the symptoms are similar to those seen in the post-diphtheritic forms of paralysis. This form of paralysis manifests itself from the second to the sixth week after the onset of the disease. In mild forms, it may begin with a paralysis of the muscles of the soft palate, which remains localized. The child has a nasal tone of voice, and liquid food is regurgitated through the nose on swallowing. In severe cases there are in addition loss of the patellar reflexes, ataxic conditions, inability to sit upright or to stand, oculomotor paralysis, facial paralysis, pallor, weak heart, arrhythmia, loss of appetite, and albuminuria.

Recovery may take place even when there is general involvement of the muscles. The great danger is extension of the paralysis to the

diphtheritic paralysis occurs in 5 to 7 per cent. of diphtheria, according to Baginsky, who reported 131 and 2300 cases of diphtheria. The soft palate was

Among the other forms of paralysis are and oculomotor nerves, the larynx (recurrent

*Bacillus diphtheriæ* alone may cause all the symptoms. In these

the pneumonia resulting from the diphtheritic or pseudodiphtheritic processes complicating scarlet fever and measles, Prudden and Northrup have shown that the *Streptococcus pyogenes* is an active causal agent. The onset of a complicating pneumonia is generally indicated by an exacerbation of the dyspnoea, fever, and cough. The prostration is also more marked. Auscultation of the inferior lateral or posterior parts of the chest on one or both sides reveals the presence of bronchopneumonia; while resolution is taking place in one part of the lung, other areas are being involved. Thus an apparent improvement may be followed by a rapid rise of temperature, increased dyspnoea, and rapid pulse. This form of bronchopneumonia may be complicated by pleuritis of a serous, serofibrinous, purulent, or hemorrhagic type.

**Gastro-enteritis.**—In nurslings there is frequently a diarrhoea with green stools and vomiting. In some cases these symptoms may become severe. Extension of the membrane into the œsophagus, stomach, and gut may take place, with a fatal result. The cases of simple diarrhoea are directly due to the swallowing of bacteria from the mouth and fauces. The diarrhoea may be so severe as to become one of the leading features of the disease.

**Retropharyngeal abscess** occurs in the tonsillar and pharyngeal forms of diphtheria as a result of infection of the retropharyngeal lymph-nodes by streptococci.

**Nephritis** may be absent, slight, or severe. Baginsky found it present in 42 per cent. of his cases. In the majority of cases of even mild diphtheria there is albuminuria; in some the urine may, in addition, contain casts, blood-cells, renal epithelium, and leucocytes, showing grave lesions of the kidneys.

**Heart paralysis.** Of greatest clinical significance is the cardiac diphtheritic paralysis, which may become apparent either early during the course of the disease or later on in convalescence. The early form may set in while the membrane is still visible in the throat. It occurs in the septic forms of the disease. These are the severe cases. The children show great prostration and apathy; the pulse is rapid and irregular; the heart-sounds, especially the muscular sound, is indistinct; the pulse is feeble and flickering; there are vomiting and abdominal pain. These symptoms may repeat themselves in attacks, until finally the patient dies with all the symptoms of collapse, such as cool extremities and shallow respirations. In such cases there is, as a rule, a marked nephritis. In the late cases the symptoms of cardiac failure appear from the *second week of the disease to the seventh week of the convalescence.* *The membrane has disappeared from the throat. There may be no premonitory symptoms, or there may have been a slight blowing* *murmur.* In their mildest form the heart symptoms



class occur as a complication of diphtheria elsewhere in the body or

and the muscular sound is weak; the pulse becomes small and either slow or rapid (tachycardia). There may be attacks of syncope, during which the patients vomit, complain of abdominal pain, and refuse medicine and nourishment. Sudden cardiac failure and death without symptoms, premonitory or otherwise, may occur in the period of convalescence.

Mild forms of cardiac irregularity which do not eventually prove fatal are seen in the beginning of convalescence. The severe forms of cardiac paralysis set in with symptoms of the early cases. These symptoms may have been preceded by the milder symptoms of cardiac irregularity. There is slight albuminuria. Suddenly, while in apparent good health, the patients complain of dyspnoea and pain in the stomach. The lips become cyanosed and the extremities cool, the pulse thready, the heart impulse weak, the heart-sounds scarcely audible; the heart may be rapid or as slow as 40 to 50 beats per minute. Vomiting is repeated, and in some cases the liver is enlarged. The patients may survive one or two such attacks, only to succumb finally. In the early forms of cardiac paralysis there may be no gross lesions in the heart-muscle. In the later forms the lesions are more apparent. There are fatty parenchymatous changes. In other cases there may in addition be changes in the vagi.

**Diphtheritic Paralysis.**—Paralyses are the result of the action of the toxins of the *Bacillus diphtheriæ* on the nerve-trunks and tissues of the general nervous system. The paralysis may occur in the course of the disease or during convalescence. When the paralysis occurs early, it affects the *velum pendulum palati*. In cases which result fatally the heart becomes affected, pneumonia caused by the passage of food into the larynx develops, or the paralysis may become general. In the latter case the symptoms are similar to those seen in the post-diphtheritic forms of paralysis. This form of paralysis manifests itself from the second to the sixth week after the onset of the disease. In mild forms, it may begin with a paralysis of the muscles of the soft palate, which remains localized. The child has a nasal tone of voice, and liquid food is regurgitated through the nose on swallowing. In severe cases there are in addition loss of the patellar reflexes, ataxic conditions, inability to sit upright or to stand, oculomotor paralysis, facial paralysis, pallor, weak heart, arrhythmia, loss of appetite, and albuminuria.

Recovery may take place even when there is general involvement of the muscles. The great danger is extension of the paralysis to the diaphragm. Post-diphtheritic paralysis occurs in 5 to 7 per cent. of the cases of diphtheria, according to Baginsky, who reported 131 cases of paralysis in 2300 cases of diphtheria. The soft palate was most often affected.

*Bacillus diphtheriæ* alone may cause all the symptoms. In these cases not only the toxins, but the bacillus itself also enters the circulation. The pharynx, tonsils, and nares are covered with a dirty brown or greenish membranous exudate. There is an ichorous discharge from the nares. The tonsils, pharynx, and lymph-nodes of the neck become necrotic. The membrane is discharged from the nose and mouth. The fetor of the breath is extreme, and the prostration correspondingly great. The larynx, trachea, and lungs may be involved in the diphtheritic process. The pulse is weak and rapid. The temperature may not be above the normal, and in some cases may be subnormal. Acute nephritis may be present. In some cases hemorrhage under the skin and from the nose, mouth, bowel, and even kidney, may precede death.

A few cases recover, but in them the necrosis of tissue in the pharynx and larynx causes permanent defects and cicatricial contractures. Loss of the uvula and perforations of the soft palate may result from diphtheria in early life.

**Laryngeal diphtheria** (*croup*) is the result of the extension of a mild or severe tonsillar or pharyngeal diphtheria. There may be no preceding clinical manifestations. There are the rare cases of so-called ascending croup, whose existence has not been wholly disproved. Cases are seen in which the most careful inspection has failed to detect preceding disease of the pharynx, epiglottis, or tonsils. Lastly, there is a class of cases which occurs during convalescence from pharyngeal or tonsillar diphtheria.

The symptoms vary accordingly as the disease manifests itself first in the larynx or follows a localized tonsillar or pharyngeal diphtheria. In the latter case there may be slight redness of the tonsils or pharyngeal mucous membrane, or the parts above the larynx may show membranous deposits. In either case the laryngeal invasion is ushered in by croupy cough and stridulous or metallic breathing. The cough is harassing and persistent, and the stridor increases within twenty-four or forty-eight hours to such an extent as to be distinctly audible, and to give the impression that there is a mechanical obstruction in the larynx. The breathing becomes labored, and there is retraction of the parts above the sternum and of the peripneumonic groove, especially at the epigastrium. In rachitic infants the sides of the chest and the epigastrium are markedly retracted at each descent of the diaphragm. With increasing obstruction the face assumes an anxious expression, the lips become cyanosed, and the surface cool. The pulse is rapid—120 to 180. The fever may be high or low. The lividity of the face in the severer forms of dyspnoea gives place to pallor. The picture of laryngeal obstruction, with the stridulous breathing, increased respirations, and overaction of the accessory muscles of respiration, is so characteristic as to be significant to even the inexperienced



observer. During the paroxysms of coughing membranous casts are expelled from the larynx. The membrane may extend downward, involving the trachea and bronchi, casts of which may be expelled. The lungs may become involved, and in severe cases are the seat of a bronchopneumonia of streptococcic nature. With this there may be compensatory emphysema. The urine may show the existence of slight or extensive nephritis, or may be normal in every respect.

Especially deceptive are those cases of membranous laryngeal diphtheria or croup whose onset closely resembles that of so-called catarrhal laryngitis. In these the symptoms may develop suddenly, and within twenty-four hours the patient presents all the symptoms of laryngeal obstruction (croup d'emblée of the French). Inspection may show little variation from the normal appearances in the pharynx. We should be cautious not to assume that no membrane is present in the larynx. Cases have been recorded in which laryngoscopic examination failed to show membrane in the larynx, but in which post mortem it was found present beneath the cords and in the trachea.

**Course and Duration.**—In the mildest and purely local forms the disease reaches its height in from two to four days; the temperature then drops to the normal and convalescence is established. In the severe septic forms the membrane spreads from the tonsils to the pharynx, and the disease attains its full development in from five to eight days. The temperature falls by lysis or crisis, and convalescence is established. If the case is very severe, the disease shows no tendency to limit itself, the toxæmia is extreme and the involvement of the lymph-nodes is very great. Death may ensue in from a week to fourteen days. In some very malignant cases death may ensue in from three to four days after the onset of the disease. The laryngeal diphtheritic croup reaches its full development as a rule early—within three days. The disease may then retrograde under treatment or may advance into the trachea and bronchi, and cause death in a variable length of time.

The **complications** include bronchopneumonia, pleuritis, gastro-enteritis, retropharyngeal abscess, suppuration or necrotic destruction of the lymph-nodes of the neck, nephritis, cardiac paralysis, early and late (or post-diphtheritic) general paralysis, and diphtheria of the eyes, skin, and vulva.

**Bronchopneumonia and Pleuritis.**—Bronchopneumonia is found in from 50 per cent. (Baginsky) to 80 per cent. (Talamon) of the autopsies on children who have died of diphtheria. It results from extension of the disease from the trachea into the smaller bronchi and alveoli of the lung, and is therefore always a true bronchopneumonia. Through the investigations of Löffler, Flexner, Northrup, and Prudden, it has been proved that the diphtheria bacillus, the *Streptococcus pyogenes*, the *Staphylococcus pyogenes*, and the pneumococcus are the exciting causes of the pneumonia. In

the pneumonia resulting from the diphtheritic or pseudodiphtheritic processes complicating scarlet fever and measles, Prudden and Northrup have shown that the *Streptococcus pyogenes* is an active causal agent. The onset of a complicating pneumonia is generally indicated by an exacerbation of the dyspnoea, fever, and cough. The prostration is also more marked. Auscultation of the inferior lateral or posterior parts of the chest on one or both sides reveals the presence of bronchopneumonia; while resolution is taking place in one part of the lung, other areas are being involved. Thus an apparent improvement may be followed by a rapid rise of temperature, increased dyspnoea, and rapid pulse. This form of bronchopneumonia may be complicated by pleuritis of a serous, serofibrinous, purulent, or hemorrhagic type.

**Gastro-enteritis.**—In nurslings there is frequently a diarrhoea with green stools and vomiting. In some cases these symptoms may become severe. Extension of the membrane into the oesophagus, stomach, and gut may take place, with a fatal result. The cases of simple diarrhoea are directly due to the swallowing of bacteria from the mouth and fauces. The diarrhoea may be so severe as to become one of the leading features of the disease.

**Retropharyngeal abscess** occurs in the tonsillar and pharyngeal forms of diphtheria as a result of infection of the retropharyngeal lymph-nodes by streptococci.

**Nephritis** may be absent, slight, or severe. Baginsky found it present in 42 per cent. of his cases. In the majority of cases of even mild diphtheria there is albuminuria; in some the urine may, in addition, contain casts, blood-cells, renal epithelium, and leucocytes, showing grave lesions of the kidneys.

**Heart paralysis.** Of greatest clinical significance is the cardiac diphtheritic paralysis, which may become apparent either early during the course of the disease or later on in convalescence. The early form may set in while the membrane is still visible in the throat. It occurs in the septic forms of the disease. These are the severe cases. The children show great prostration and apathy; the pulse is rapid and irregular; the heart-sounds, especially the muscular sound, is indistinct; the pulse is feeble and flickering; there are vomiting and abdominal pain. These symptoms may repeat themselves in attacks, until finally the patient dies with all the symptoms of collapse, such as cool extremities and shallow respirations. In such cases there is, as a rule, a marked nephritis. In the late cases the symptoms of cardiac failure appear from the second week of the disease to the seventh week of the convalescence. The membrane has disappeared from the throat. There may be no premonitory symptoms, or there may have been a slight blowing murmur at the apex. In their mildest form the heart symptoms appear in the second or third week. The heart becomes irregular,



and the muscular sound is weak; the pulse becomes small and either slow or rapid (tachycardia). There may be attacks of syncope, during which the patients vomit, complain of abdominal pain, and refuse medicine and nourishment. Sudden cardiac failure and death without symptoms, premonitory or otherwise, may occur in the period of convalescence.

Mild forms of cardiac irregularity which do not eventually prove fatal are seen in the beginning of convalescence. The severe forms of cardiac paralysis set in with symptoms of the early cases. These symptoms may have been preceded by the milder symptoms of cardiac irregularity. There is slight albuminuria. Suddenly, while in apparent good health, the patients complain of dyspnoea and pain in the stomach. The lips become cyanosed and the extremities cool, the pulse thready, the heart impulse weak, the heart-sounds scarcely audible; the heart may be rapid or as slow as 40 to 50 beats per minute. Vomiting is repeated, and in some cases the liver is enlarged. The patients may survive one or two such attacks, only to succumb finally. In the early forms of cardiac paralysis there may be no gross lesions in the heart-muscle. In the later forms the lesions are more apparent. There are fatty parenchymatous changes. In other cases there may in addition be changes in the vagi.

**Diphtheritic Paralysis.**—Paralyses are the result of the action of the toxins of the *Bacillus diphtheriae* on the nerve-trunks and tissues of the general nervous system. The paralysis may occur in the course of the disease or during convalescence. When the paralysis occurs early, it affects the *velum pendulum palati*. In cases which result fatally the heart becomes affected, pneumonia caused by the passage of food into the larynx develops, or the paralysis may become general. In the latter case the symptoms are similar to those seen in the post-diphtheritic forms of paralysis. This form of paralysis manifests itself from the second to the sixth week after the onset of the disease. In mild forms, it may begin with a paralysis of the muscles of the soft palate, which remains localized. The child has a nasal tone of voice, and liquid food is regurgitated through the nose on swallowing. In severe cases there are in addition loss of the patellar reflexes, ataxic conditions, inability to sit upright or to stand, oculomotor paralysis, facial paralysis, pallor, weak heart, arrhythmia, loss of appetite, and albuminuria.

Recovery may take place even when there is general involvement of the muscles. The great danger is extension of the paralysis to the diaphragm. Post-diphtheritic paralysis occurs in 5 to 7 per cent. of the cases of diphtheria, according to Baginsky, who reported 131 cases of paralysis in 2300 cases of diphtheria. The soft palate was most often affected. Among the other forms of paralysis are those of the facial and oculomotor nerves, the larynx (recurrent laryngeal), and lastly forms of ataxia. Antitoxin has little effect in

preventing these paralyses. They occur as frequently after its administration as during the pre-antitoxin period.

In the American Pediatric Society's tabulation 9.7 per cent. of the cases had paralysis; of these, 32 out of a total of 328 cases died of cardiac paralysis.

Hemiplegic cerebral palsy may occur in diphtheria (Monti, Levi, Baginsky).

**Disturbances of the sensory nerves** also occur in diphtheria, such as perversions of the senses of smell and taste; also anæsthesia of the rectum.

**Psychical derangements** such as melancholia, have been reported.

**Diphtheritic Ophthalmia.**—True diphtheritic ophthalmia occurs both as an accompaniment of diphtheria of the fauces and as an idiopathic affection. There are two distinct forms of pseudomembranous affection of the eye. In the first, the Löffler bacillus is present, but in the second, or diphtheroid form, it is absent, and the streptococcus alone is found. Of the true diphtheritic form, one class of cases has a mild clinical course. In these the bacillus isolated resembles the pseudodiphtheria bacillus in not possessing virulent properties. In the other form of diphtheritic eye affection the membrane spreads rapidly and causes destruction of the eye. The diphtheritic invasion is ushered in with redness and chemosis. The membrane appears first on the palpebral conjunctiva, and causes marked swelling of the lids. There is little seropurulent discharge. In the progressive form destruction and perforation of the cornea result. I have seen several cases in connection with fatal diphtheria complicating measles, and also cases in which there was no history of diphtheria in the patient or family. I have seen it occur as an idiopathic affection in nurslings. According to Baginsky, diphtheritic ophthalmia occurs in 3 per cent. of the cases of diphtheria, and is most frequent from the second to the sixth year.

**Diphtheria of the skin** occurs when the specific bacillus finds lodgement in an abrasion or cut. The membrane spreads over the wound and encroaches on the surrounding skin.

**Diphtheria of the vulva** is met with both as an idiopathic affection and as a complication of true diphtheria elsewhere in the body. I have not found the Klebs-Löffler bacillus in a number of pseudomembranous inflammations of the vulva and vagina in infants. Some of these cases show the presence of true membrane; others begin as aphthous ulceration and develop membrane later. These cases are benign. The diphtheritic bacillary cases may be divided into two distinct classes according to their causation. The cases of one class show the Löffler bacillus, but are benign in course, although I have proved by animal experiment the presence of the bacillus of diphtheria in virulent form. In the other class of cases there is extensive destruction of tissue, and sometimes a fatal result. Cases of this



class occur as a complication of diphtheria elsewhere in the body or in connection with the exanthemata.

The symptoms of diphtheria of the vulva and vagina may be localized strictly to the parts, or there may, as in the severer forms of Henoch, be constitutional symptoms of toxæmia. Locally, the disease is characterized by the appearance of patches of membrane on the inner surface of the labia, clitoris, and introitus vaginae. The parts, especially the labia majora, are intensely swollen and œdematous. In Henoch's cases there was gangrene or necrosis of neighboring tissues. In my cases there was no complicating diphtheria of other parts. The cases occurred in infants and in children under two years. They were benign in course, although of bacillary type.

**Nasal Passages.**—Councilman, Mallory, and Pearce, in their latest monographs on diphtheria, call attention to the frequency of invasion of the accessory sinuses of the nose and antrum by the diphtheritic process. They found the antrum affected in 33 cases of 52 examined. Clinically, this affection is more common than appears from these figures. This would account, according to these authors, for the persistence with which diphtheria bacilli continue in the nasal secretions after the throat lesions have disappeared. The disease of the antrum may, as pointed out by Wolff, and recently by Mayer, persist after the diphtheria has run its course. Mayer classifies the symptoms as eversion of the lower lid, fistulous opening in the cheek from which pus exudes, and a fetid purulent discharge from the nose on the side of the face at which the fistula is situated.

**Other Complications.**—Diphtheria in *pertussis* is a serious complication, since the resistance of the patient is generally much decreased. *Bronchopneumonia* is especially to be feared. In *tuberculosis* the patient usually dies as a direct result of the complication. In *measles* the diphtheritic process is a grave complication; it may invade the larynx and death may ensue from extension of the disease to the lungs. In *typhoid fever* the process causes death by invasion of the lungs.

**Exanthem.**—Is there an exanthem characteristic of diphtheria? I am inclined to view all eruptions which may occur in the course of this disease as purely accidental. They may be the result of remedies (antitoxin) administered or of some infection originating in the gut. Among these eruptions are the various forms of erythema and roseola. Erythema urticatum is often seen.

The **diagnosis** of diphtheria must be considered in its clinical and bacteriological aspects. Clinically the characteristic and ever-present lesion is the membrane. This is seen on the tonsils, uvula, pillars of the fauces, and the posterior pharyngeal wall. Its color varies. In consistency it may vary from a thin pellicle or cloudy discoloration to a thick adherent, pultaceous or stringy mass. In a large proportion of cases the presence of the membrane and other characteristics are presumptive evidence of diphtheria. On the

other hand, there are certain forms (not very frequent) of pseudo-membranous inflammation of the tonsils and fauces which are not truly diphtheritic; these are called pseudodiphtheria or diphtheroid. In these cases the Klebs-Löffler bacillus is not found, but streptococci, staphylococci, and other bacteria are present. Some forms of diphtheria show at first only fibrinous specks on the tonsils; in others there are small necrotic ulcerations on the tonsil, and in still others the diphtheria may simulate an acute catarrhal follicular amygdalitis or lacunar amygdalitis. These cases are not as infrequent as was formerly supposed. In the pseudomembranous and other forms of inflammation of the throat above described a bacteriological test should always be made. It should be practised as a routine procedure in all cases of angina. Cultures should be made in cases of laryngeal inflammation in which no membrane is visible in the fauces. If membrane be present in the fauces, and a culture fail to reveal the Klebs-Löffler bacillus, a second and even a third culture should be made. I have frequently established the presence of the specific bacillus in membrane in cases in which the first culture-test proved negative. It is not a reliable nor satisfactory method to spread membrane or secretion from the throat direct on a cover-glass, and decide from such a preparation the nature of the process. The technique of culture-tests is scarcely within the scope of this work. It is sufficient to state that growth can be obtained within four or five hours if the culture-tube is subjected to a temperature of  $100.4^{\circ}$  to  $102.2^{\circ}$  F. ( $38^{\circ}$  to  $39^{\circ}$  C.) in a small incubator. Other diseases, such as membranous forms of stomatitis, may simulate diphtheria. In these cases the culture test is the only positive mode of making a diagnosis. Certain forms of laryngismus stridulus resemble acute diphtheritic laryngitis, or a diphtheritic process may be present in the larynx in a rachitic infant subject to attacks of laryngismus. Cultures should be made in all such cases.

In small towns and country districts the practitioner without the aid afforded by laboratories will often be thrown on his own resources in making a diagnosis. In such cases the following clinical symptoms may be considered fairly presumptive evidence of diphtheria:

The presence of membrane on a tonsil and a small patch, streak, or speck of membrane on the adjacent surface of the uvula or tip of the uvula; a patch of membrane on the tonsil and an accompanying patch on the posterior pharyngeal wall; the presence of a croupy cough and stridulous breathing with small patches of membrane on the tonsil or epiglottis, are all of much diagnostic value. The presence of albumin in the urine is of little value in making a diagnosis, as it may be present in non-diphtheritic affections and absent in diphtheria. Constitutional symptoms are only of corroborative value. It is well known that the most virulent forms of diphtheria may at first be manifested by few constitutional symptoms. The



temperature-curve is not characteristic. If a patient who at first suffers from a catarrhal tonsillitis or pharyngitis, shows within twenty-four hours minute patches of membrane either on the uvula or pharynx, it may reasonably be assumed that true diphtheria is present. An acute laryngeal inflammation, croupy cough, and stridulous breathing which not only persist beyond the first twenty-four hours or first night, but also become aggravated, justify a diagnosis of diphtheria of the larynx, although no membrane is visible in the throat. General symptoms are of little diagnostic value. Rhinitis at first accompanied by a serous and later by a fetid sanguinolent discharge, with glandular swellings in the neck, is diagnostic of diphtheria.

Adenitis is frequently absent at the outset of tonsillar diphtheria, even when patches of membrane of some size are present. On the other hand, a simple catarrhal tonsillitis is often accompanied by marked adenitis.

Paralysis of the soft palate, appearing in the course of a severe or mild pseudomembranous tonsillar, pharyngeal, or laryngeal inflammation, or after the affection has run its course, points strongly to true diphtheria, although cases of paralysis of the soft palate following diphtheroid have been reported. The color of the membrane, its detachability, and the fact that a bleeding surface is left after its removal, cannot be relied upon as aids to diagnosis, in view of the fact that interference with the membrane is not advisable.

Aphthæ with pseudomembrane over the vault of the hard palate, spreading to the gums and cheeks, are seen in newly born and older infants. These forms of pseudomembranous stomatitis are the result of traumatism inflicted by the infected fingers of the nurse or mother, and are limited to the parts on which they are first seen. Such septic membranes rarely spread unless the exciting causes are perpetuated.

Herpes of the pillars of the fauces, so-called herpes of the tonsils, has often in my clinic been mistaken for diphtheritic patches. With a suitable light such an error should seldom be made.

Following the ingestion of caustic alkali or the traumatism consequent on washing or rubbing the mucous membrane, aphthous ulcerations, which closely simulate diphtheritic membranous patches, are prone to appear over the hamular process of the palate bone. The history of the case, the absence of diphtheria elsewhere, and the result of a culture test will exclude diphtheria.

The patches of necrotic tissue seen on the tonsils, pillars of the fauces, and uvula following tonsillotomy and ablation of adenoids, and sometimes accompanied with paralysis, may mislead the observer and cause him to make a diagnosis of true diphtheria.

The membranous patches which appear on the tonsils of scarlet fever patients at the outset of the disease are for the most part diphtheroid. Unless the patient has been exposed to a double infection, which is infrequent in private practice, the patches of membrane

which appear later in the disease are also of a diphtheroid nature. True diphtheria may coexist with scarlet fever (Baginsky, Escherich, Councilman), but does so in only a small number of cases.

The appearance of a pseudomembranous exudate on the tonsils of a patient attacked with measles should be regarded as diphtheritic until the contrary has been proved. The laryngitis with croupy cough and breathing often complicating measles is not, as a rule, diphtheritic.

The **prognosis** and mortality vary with the age of the patient, the form and severity of the infection, and the extent to which organs other than the fauces and larynx are involved. Young infants, unless coming early under observation, give a high mortality rate. Septic forms of diphtheria are more fatal than those in which the process is a distinctly local affection. The mortality also varies with the nature of the epidemic. In Baginsky's statistics of 2711 cases, the mortality from the sixth to the twelfth month was 52 per cent.; from the second to the third year, 37 per cent., decreasing to 8 per cent. in the tenth year. The death-rate is high in infants and children of delicate constitution and in those suffering from any form of dyscrasia.

The **treatment** of diphtheria may be prophylactic, constitutional, and local.

**Prophylaxis.**—The patient should be isolated as soon as the membranous deposit is detected. Other children of the family who have been in contact with the patient should at once be given immunizing doses of antitoxin, and the furniture of the sick-room, such as hangings and carpets, should be removed, only the most necessary articles being retained. The room should be well ventilated. The nurse should not come in contact with other members of the family. All articles of clothing worn by the patient should be dipped in an antiseptic solution (corrosive sublimate, 1:2000) before removal from the sick-room. The physician, before entering the sick-room, should cover his head with a cap and wear a long coat or bath-robe, which should be hung outside the sick-room. If it is necessary for members of the family to enter the room, they should observe the same precautions, and on leaving the room they should gargle or rinse the mouth with some mild cleansing solution, preferably of boric acid. A culture should at once be made. The swab should be rubbed over the tonsils if they are the seat of exudate; if the case is laryngeal, the swab is passed over the epiglottis and posterior pharyngeal wall. Utensils used in feeding the patient should not be used by others.

The patient after convalescence should not mingle with other children until culture has proved the absence of the *Bacillus diphtheriæ* from the throat.

**Constitutional treatment** consists first in the administration of diphtheria antitoxin. It is not within the scope of this work to enter into the details of the theory of action of this agent, which is



the outcome of the modern experimental method of the investigation of disease. Its place in the therapy of diphtheria is now assured. The mortality of diphtheria has been greatly reduced since its introduction. Baginsky gives the following figures, showing the mortality before and after the introduction of antitoxin :

Age.	Before.	After.
Two years . . . . .	60.2 per cent.	25.8 per cent.
Two to four years . . . . .	51.2 " "	17.1 " "
Eight to ten years . . . . .	28.8 " "	10 " "

Of 5794 cases in private practice collected by the American Pediatric Society, the total mortality was only 12.3 per cent. In the cases injected on the first day of the disease the mortality was 7.3 per cent. In the laryngeal form of diphtheria the results have been especially favorable. In 1704 cases operated and not operated there was a mortality of 21 per cent., of the intubated cases, 23 to 27 per cent., as against 60 to 70 per cent. before the introduction of antitoxin.

The DOSAGE varies with the age of the patient, the severity of the infection, and the duration of the case before the beginning of treatment. Mild forms of local membranous affections of the tonsils and pharynx coming under observation on the first day should receive doses of antitoxin as follows: Up to one year, 600 units; one to two years, 1000 units; two to five years, 1500-2000 units. If the disease has markedly progressed twenty-four hours after the first injection, the initial dose should be repeated. The severer forms of localized diphtheria with marked constitutional symptoms should receive initial doses half as large again or twice as large. Laryngeal forms should receive proportionately large doses. The American Pediatric Society recommends as an initial dose 1500 units for a child under two years, and 2000 units for one above that age. I employ 300 units for immunizing purposes in very young infants, and 500 units in older children.

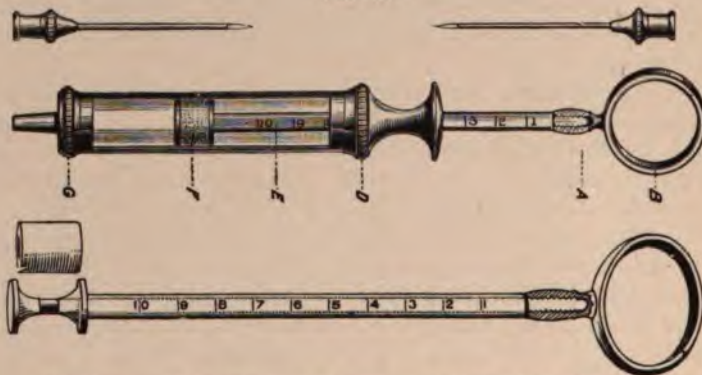
The immunizing power extends over a period of three weeks. It is best to give an initial dose of sufficient amount, so that a repetition of the dose will not be necessary; on the other hand, it is advisable not to give an excessively large dose. The concentrated antitoxins are preferable both on account of the diminished bulk and the infrequency with which skin and joint-affections follow their injection. Recently prepared antitoxin should be obtained, for it has been shown that this agent deteriorates with age (Abbott), and then no longer contains the original unit values.

TIME OF INJECTION.—The antitoxin should be given as early in the course of the disease as possible. If membrane is present, no time should be lost in waiting for the result of the culture test, for if the disease is not true bacillary diphtheria no harm can result from the injection, while to wait may be hazardous to the patient.

**MODE OF INJECTION.**—The syringe with asbestos packing should be used for making injections. Such an instrument is easily cleansed and boiled. I find the back just above the buttock the most convenient location in which to inject. The child can be easily held if this site is chosen. The parts should be carefully cleansed. The injection is given in the same manner as a hypodermic injection. The parts should not be rubbed after the injection, and the puncture should be covered with a small square of iodoform gauze.

**EFFECT OF INJECTION.**—There is a slight temporary rise of temperature following the injection. It is thought to be due to the entrance into the blood of the additional toxin contained in the antitoxin. This rise is succeeded by a gradual or critical fall, which continues until the temperature is subnormal. The membrane ceases

FIG. 53.



Antitoxin syringe with asbestos packing; can be taken apart and sterilized.

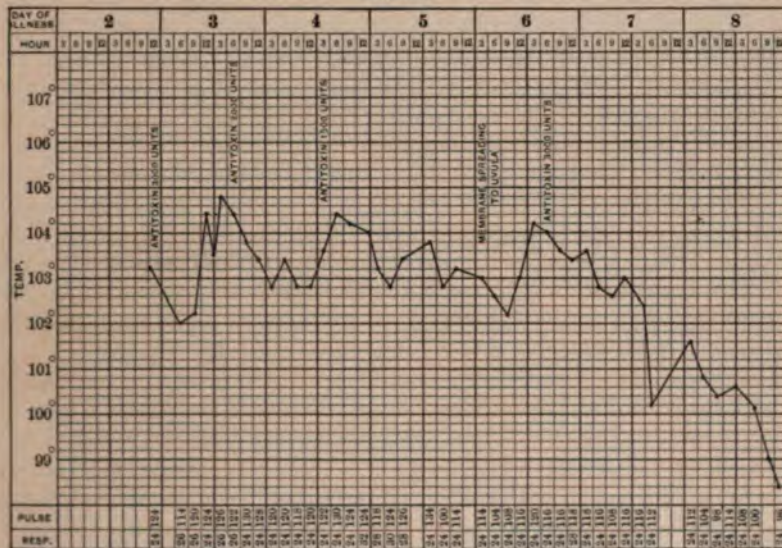
to spread and exfoliates. In some cases these phenomena may be delayed twenty-four hours. The next day the pulse drops, the prostration gives way to a clear sensorium and good heart action, and sometimes the children sit up in bed and play with toys. The glandular swelling also diminishes markedly. In laryngeal cases if there has been threatened stenosis, the symptoms retrograde. Of 258 cases of this kind, Baginsky saw fully one-half retrograde spontaneously. On the other hand, if the temperature persists high after twenty-four hours and the membrane continues to spread, the injection should be repeated, especially if the swelling of the lymph-nodes is marked and there are symptoms of septic infection.

The effect of an injection of antitoxin on the blood is to diminish the number of leucocytes; just prior to the fall of temperature there is a critical hyperleucocytosis (Ewing, Schlessinger). Albuminuria continues, but this is also the case not only when no antitoxin has been used, but also in almost any infectious disease in which bacteria or their toxins circulate in the blood.



The eruptions which occur after the injection of antitoxin are of interest. At the site of the injection an abscess or phlegmon may form. This is the result of uncleanness in technique or is due to some irritating substance in the antitoxin. A brawny erythema which gradually disappears may appear in a day or more at the site of injection. The injection may be rapidly followed by a painful eruption on the extremities, consisting of circumscribed violet colored spots, closely resembling erythema nodosum. The subcutaneous tissues are swollen, the joints are painful, and in addition there may be elevated temperature and a cardiac murmur. Herpes labialis and herpes nasalis, urticaria-like general eruptions, and morbilliform

FIG. 54.



Septic form of tonsillar diphtheria; both tonsils and soft palate involved with nasopharynx. Persistence of temperature and recurrence of membrane after antitoxin injections on the sixth day. Injection of additional antitoxin, and critical drop of temperature thereafter. Recovery. Boy, six years of age.

or scarlatiniform eruptions have followed injections. These eruptions appear from a few days to fourteen days after the injection.

Conjunctival injection, tachycardia, and arrhythmia may be present.

The acute symptoms described above subside in most cases within two or three days.

Kidney irritation may follow the injection of large doses of antitoxin. In many of the cases reported, however, the renal symptoms have not been due to the antitoxin alone, and the same may be said of the recorded cases of endocarditis following antitoxin injections.

The introduction of antitoxin has by no means lessened the

necessity of careful general management of a case by the physician. The temperature is controlled or modified by the ordinary hydrotherapeutic procedures. Antipyretics of the coal-tar series should not be administered, as they weaken the heart.

If signs of cardiac paralysis of the early type set in, full doses of the cardiac remedies—digitalis (if the pulse is rapid), strychnine, camphor, musk, and especially whiskey—are given. In order to guard against cardiac weakness in the later period of the disease, a cardiac stimulant, such as strychnine, is given in small doses throughout the illness and in convalescence. The patient is not allowed to sit up too early should signs of cardiac irregularity appear at the outset of convalescence. In all cases of diphtheria the utmost caution should be exercised in reference to the heart.

The infant should not be nursed at the breast, lest the breast be infected. The milk should be pumped off and fed to the infant with a bottle. If there is diarrhœa, the milk is suspended and the bowel irrigated. The milk should not be resumed until all danger from this source is past. I administer alcohol in moderate doses if the prostration, pulse, and temperature warrant it. Infants under a year should be given half a drachm (2.0) of whiskey every three hours; infants more than two years of age, a drachm (4.0) at the same intervals. Diphtheria patients, especially those suffering from the septic form with constitutional symptoms, should be kept recumbent. The administration of remedies should not be forced, for struggling on the part of the patient may prove dangerous to the heart. During convalescence the whiskey may be replaced by wine. In these cases strychnine in small doses (grain  $\frac{1}{200}$  [0.0003]) should be continued for some time, in order to support the heart. I advise a return to a mixed nutritious diet in all cases as soon as the temperature is normal; in this way the effect of the toxins on the tissues is counteracted as much as possible.

Some physicians still resort to the internal administration of corrosive sublimate in doses of grain  $\frac{1}{100}$  (0.0006) or more, according to the age of the patient. It is given in the septic tonsillar and nasal cases, and also in the laryngeal forms of diphtheria.

**Local Treatment.**—The presence of bacteria other than the diphtheria bacillus around the local lesions necessitates the use of local cleansing and disinfecting measures. In very young infants the nasal discharges are washed away by means of a glass syringe with a blunt rubber tip. The infant is laid on the side, and the nurse, standing behind the patient, irrigates the nostrils with normal salt solution at 110° F. (43.3° C.), as shown in Fig. 7. A pus basin is held underneath the chin. Older children will struggle, but by suasion they may be irrigated in the sitting posture. If there is much resistance, it is not desirable to insist on irrigation. In irrigating, the syringe should have a position parallel with the floor of the



nasal fossæ. Spraying with a mild solution of listerine or Dobell's solution is possible in some children, impracticable in others. The lymph-nodes, if slightly enlarged, are best treated by the application of warm oil of hyoscyamus; if very much swollen, the application of cloths wrung out in ice-cold water is of great utility. Small pieces of ice swallowed whole are grateful to the patient.

**Treatment of Laryngeal Diphtheria.**—In cases of mild laryngeal diphtheria an injection of antitoxin should be given. The patient should be placed under a tent, and grains x (0.6) of calomel sublimed every two or three hours, according to the necessities of the case. The efficacy of the calomel vapor is increased by passing steam into the tent at the same time. A convenient method is to place the calomel in a spoon, and heat the spoon over an ordinary candle, placed within the tent. The swelling of the larynx caused by the invasion of the *Bacillus diphtheriæ* and other bacteria is quickly relieved by the calomel, particularly in croupy cases with little or no membrane visible above the larynx. A tent may be improvised as described on page 38. Steam saturated with benzoin or thymol may also be passed into the tent. A croup kettle may be improvised from an ordinary teapot or one sold for the purpose may be employed. It is sometimes necessary to suspend the steam inhalations for an hour or longer, for the purposes of ventilation. The general treatment as to the heart, temperature, and food is the same as in the tonsillar forms of diphtheria. If signs of mechanical obstruction appear, intubation is indicated.

**INTUBATION.**—To Joseph O'Dwyer, of New York, belongs the credit of having perfected a method of relieving membranous obstruction of the larynx in diphtheria. Intubation in America and on the continent of Europe has completely displaced tracheotomy as a remedy for relieving laryngeal obstruction due to diphtheria.

**Instruments.**—Intubation tubes (Fig. 55) are of metal coated with rubber, though originally made of gilt metal. The tubes are grad-

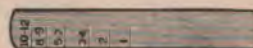
FIG. 55.



O'Dwyer tube.

uated (Fig. 56) according to the age of the patient, and in their present form are the most ingeniously devised instruments ever given by American medicine to the world. The tubes are furnished with obturators, which fit into a handle, the in-

FIG. 56.



Gauge for the age of the patient.

roducer (Fig. 57). There is, in addition, a forceps (Fig. 58) with small departing blades, called the extractor. Finally, there is a gag (Fig. 59) so constructed that it may be introduced into the mouth and kept in position without obstructing the view of the operator.

**Indications.**—We intubate when a progressive dyspnœa, which

FIG. 57.



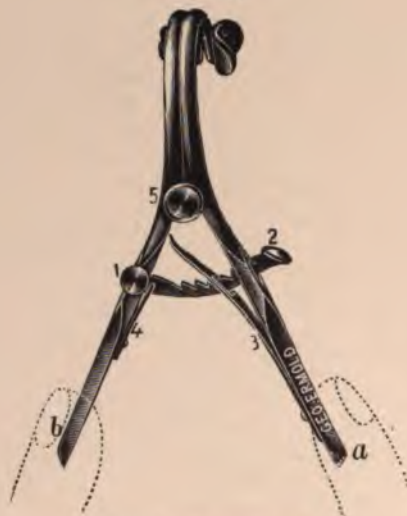
O'Dwyer tube, obturator, and handle.

FIG. 58.



The O'Dwyer extractor.

FIG. 59.



Gag of the O'Dwyer set.



FIG. 60.



Introduction of the tube along the index finger.

FIG. 61.



Passing the tube over the epiglottis.

FIGS. 60, 61.—The operation of intubation of the larynx. Position of child, operator, and assistant.

FIG. 62.



Introduction of the tube into the chink of the glottis.

FIG. 63.



The index finger pushes the head of the tube into place in the larynx.

FIGS. 62, 63.—The operation of intubation of the larynx. Position of child, operator, and assistant.

produces sensible exhaustion, exists. O'Dwyer never tubed the larynx except as a *dernier ressort*, and did not approve of early tubage. If an infant or child shows marked retraction of the supra-



sternal notch, retraction of the epigastrium, and stridor, with accompanying labored breathing, we should at once proceed to tube the larynx.

*Mode of Operating.*—The patient is wrapped in a blanket and held upright in the arms of a nurse, so that the head of the patient is on a level convenient to the operator, who stands facing the patient. An assistant standing behind the nurse steadies the head of the patient. The gag is introduced by depressing the tongue and jaw with a tongue-depressor. The assistant steadies the gag as he holds the head tilted very slightly backward. The tube, threaded with a silk ligature, is with its introducer held firmly with the right hand. The index finger of the left hand is now introduced into the mouth to the root of the tongue and search made for the epiglottis. In young infants<sup>1</sup> the epiglottis is short. The finger must be introduced quite deeply, feeling the arytenoid cartilages of the larynx, and is then drawn upward until the epiglottis is hooked forward. The index finger now holds the epiglottis (Fig. 64), and in a small

FIG. 64.



Method of hooking forward the epiglottis in intubation.

larynx a skilled operator can also feel the arytenoids (Fig. 65). The tube is now introduced in the median line of the mouth along the

<sup>1</sup> *Peculiarities of the Larynx.*—Thomson and Turner have shown that the infantile form of larynx differs materially from that found later in life. At birth and in infants and young children the epiglottis is very small and gutter-shaped. The glottis is guarded above by the aryteno-epiglottic folds, which are closely approximated to each other. Toward the tenth year the epiglottis becomes much flattened, the aryteno-epiglottic folds become widely separated, and the larynx assumes the adult type. It is important to remember these points in the operation of intubation.

palmar surface of the index finger (Fig. 60), and the finger guides the tube over the epiglottis and into the chink of the glottis and prevents its slipping into the œsophagus (Fig. 61). The instrument should always be kept in the median line. The index finger holding the epiglottis should be held well to the angle of the mouth, so as to obtain plenty of room. No force should be used, else false passages will be made. If the first attempt at introduction does not succeed, we should not persist too long, but remove the introducer rapidly and give the larynx a few moments to recover its action, and then try again. As the tube passes into the chink of the glottis the handle of the introducer is elevated, as in Fig. 62, causing the end of the instrument to lie against the base of the tongue. The tube is released, the introducer and obturator withdrawn, and the index finger gently presses the head (Fig. 63) of the tube into the larynx. The gag is withdrawn, and the silken thread passed over the ear of the patient and fixed back of the ear with a piece of rubber plaster. Some operators remove the thread after ten minutes. The advantages of leaving the thread are that, should the tube be coughed up in the absence of the physician, it can be recovered by the nurse. In extubating, it is an aid in removing the tube.

No anæsthetic is required, and ordinary assistance only is necessary. The air passing into the bronchi is moistened in its passage through the natural passages. The danger that food particles may pass into the larynx has been exaggerated. The detachment of membrane in front of the tube is very infrequent. Should it happen, and the membrane not be expelled on removal of the tube, tracheotomy is admissible if asphyxia is imminent. It sometimes happens that the tube is expelled many times after introduction. It should be reintroduced or a larger tube employed.

FIG. 65.



The infantile larynx. Its development into the adult type at the age of nine years. 1. Infant, three months of age. 2. Child, three and a half years of age. 3. Boy, nine years of age. Enlargement upward of the epiglottis and shaping of the arytenoid cartilages. (Thomson and Turner, *British Medical Journal*, December 1, 1900.)



If the operator has chosen to leave the silken cord of the tube *in situ*, it should be passed through the space between the first molar and bicuspid tooth, to avoid its being gradually bitten through. Should it be bitten through, the finger is introduced into the mouth to the top of the tube and the thread withdrawn, while the tube is kept in the larynx with the finger.

The tube is allowed to remain from twenty-four hours to five days. Since the introduction of antitoxin the tube is taken out much sooner than was formerly the practice. If there is marked improvement in two or three days, removal of the tube should be attempted and the effect of such a procedure on the breathing should be observed.

*Extubation.*—The patient is placed in the same position as for intubation. The left index finger is passed into the mouth and search made for the epiglottis, the tip of the finger resting on the arytenoids. The extractor is passed along the palmar side of the finger and is guided into the opening in the tube by the tip of the finger. Extubation is more difficult than intubation. The extractor should be regulated by means of a small screw, so that the blades do not open too far. This is to guard against injury to the soft parts of the larynx should the opening of the tube not be entered.

*Dangers.*—The dangers of intubation include detachment of membrane during introduction, laceration of the parts, the formation of false passages, and asphyxia. The first rarely occurs unless force is used. The second can only occur as a result of rough and unskilled efforts at intubation. The third occurs only following prolonged efforts at introduction of the tube. Even a skilful operator may pass the tube into the ventricle of the larynx. Northrup has published a case in which there was a false pocket above the cords which prevented the entrance of the tube into the larynx. In other cases there is what is described by O'Dwyer as subglottic stenosis. Northrup thinks that this is due to swelling of the mucous membrane at the level of the cricoid cartilage. In these cases introduction of the tube is very difficult. The operator may be compelled to use force to push the tube past the stenosis or a smaller tube may be employed. While the tube is being worn, it may become obstructed by membrane. This is denoted by a return of the croupy cough, a snarling, flapping sound, and obstruction to expiration. To obviate these difficulties, O'Dwyer has had short tubes constructed without a retaining flange. These tubes have a special introducer. The largest size for the age is chosen, and the tube forced into the larynx. These tubes should be used only by skilled operators. The tubes are allowed to remain but a short time in the larynx. Other complications are the formation of granulations or ulcerations around the lower end of the tube if it is too long, and at the cricoid cartilage if it is too large. The former condition is not serious; the latter may

destroy the cartilage. Granulations may form about the head of the tube. In this case tubes with built-up heads are used to press on the granulations, thus causing them to atrophy (Fig. 66).

*Feeding the patient* after introduction of the tube requires care. Most infants will nurse with the tube in the larynx. In some there

FIG. 66.



Built-up tubes.

is considerable difficulty in swallowing. The patient is taken in the lap of the nurse and fed with the head held a little lower than the body. Fluids thus cannot enter the trachea and cause pneumonia. This method of feeding was suggested by Casselberry, of Chicago.

**Treatment of the Complications.**—The treatment of the BRONCHO-PNEUMONIA which complicates diphtheria is similar to that employed in the treatment of the primary affection. The question of the further administration of antitoxin always arises in these cases. I give it in full doses, since it is known that the *Bacillus diphtheriæ* is the causative factor.

The GASTRO-ENTERITIS which complicates diphtheria is apt to prove a very serious complication. It should receive the same treatment

as a primary gastro-enteritis.

Both the severe and the mild cases of VULVOVAGINITIS should be treated with antitoxin. In some of the mild forms of undoubted bacillary origin which I have seen, the membrane was easily removable. In these cases, contrary to the practice in the tonsillar cases, I remove the membrane with a spud wrapped with cotton. The bleeding surface left after removal is painted with a 10 per cent. solution of silver nitrate once daily. I have cured cases by this method alone. If there are extensive swelling, necrosis, and gangrene, this method will be of no avail, and antitoxin should be given in full doses, and repeated according to indications.

**PARALYSES.**—The treatment of diphtheritic and especially post-diphtheritic paralyses is at present largely empirical. The symptoms appear with the degenerations in full progress. Of all the remedies recommended, Fowler's solution in tonic doses has seemed to give the best results. I have seen patients recover when given arsenic, nutritious food, and abundant fresh air. Hypodermic injections of strychnine are of questionable value. Electricity is of value as an adjuvant to massage of the muscles only in general paralysis. It is questionable whether in some cases it is not capable of doing great harm by tiring nerve and muscle. I find that patients do very well with hydrotherapy and manual massage. In these cases the last reaction to reappear is the patellar reflex.



**Diphtheroid.**

(*Pseudodiphtheria*; *False Diphtheria*.)

The term diphtheroid includes all pseudomembranous formations not caused by the Klebs-Löffler bacillus. It was first proposed in 1860 by Boussage, and has recently been adopted by Weigert, Escherich, Heubner, and Behring.

**Occurrence.**—This form of pseudomembranous formation is most frequently met with in the exanthemata, especially scarlet fever and measles. In the former it is a common complication. It is also met in other conditions, and may occur as a primary affection.

**Etiology.**—The cases met in the exanthemata were first described by Prudden, who believed that the process was due to a streptococcus, the *Streptococcus diphtheriae*. Since then, the occurrence of the streptococci has been confirmed, but there have also been added to this group of pseudomembranous inflammations cases in which the pseudomembrane is caused by a diplococcus, the so-called Roux coccus. The pneumococcus (Jaccoud and Menetrier) may also cause a pseudomembranous angina. The *Bacterium coli* and the gonococcus (the latter in newly born infants) may cause a membranous formation in the mouth and throat. The *Staphylococcus pyogenes aureus* is also found in these diphtheroid membranes. By far the most important group is that first mentioned, the pseudomembranous or diphtheroid inflammation caused by the *Streptococcus pyogenes*, which is none other than that isolated by Prudden. These cases are characterized by their favorable course; while the mortality in diphtheria varies from 20 to 35 per cent., according to the age of the patient, the virulence of the epidemic, and the early administration of antitoxin, the mortality of the diphtheroid cases ranges from 3 to 5 per cent. (Park, Baginsky).

**Symptoms and Course.**—The pseudomembrane occurs on the tonsils, pharynx, and larynx. There are adenopathy and fever. The prostration and constitutional disturbance are much less than in true diphtheria. Membranes and casts of the larynx and trachea may be expelled. Suppuration of the lymph-nodes may also occur. In many of these cases there is a complicating bronchopneumonia of the streptococcus type (Prudden and Northrup), which usually results fatally.

**Diagnosis.**—It is not possible to make a diagnosis of diphtheroid from the gross appearance of the membrane. The culture-test is the only reliable method of determining the nature of a pseudomembranous exudate. If the first culture gives a negative result, a second one should be made.

**Treatment.**—Clinically the treatment is much the same as in true diphtheria. The administration of antitoxin should not be delayed until the nature of the exudate is determined. It is then

discontinued. An exception to this rule may be made in the scarlatinal form of diphtheroid, in which it is safe to wait for the result of the culture-test, unless it is known that the patient has been exposed to diphtheritic infection. In such a case antitoxin should be administered. In laryngeal obstruction the indications for treatment are the same as in true diphtheria.

### SCROFULA or SCROFULOSIS.

Scrofulosis is a constitutional dyscrasia which occurs chiefly in childhood and youth, and is characterized by enlargement of the lymph-nodes and slow, sluggish inflammation of the mucous membranes, skin, joints, and bones. There is a distinct tendency to repeated inflammations from very trivial causes.

**Occurrence.**—Scrofulosis is almost exclusively a disease of childhood and youth, and is rarely seen after the twentieth year. Henoch and Birch-Hirschfeld state that the majority of cases occur between the third and the fifteenth year. Females are more frequently affected than males. Ruhl found it to be most common between the sixth and the tenth year.

**Forms.**—Cornet and Ponfick recognize three forms of scrofulosis :

*a.* The tuberculous form, which is practically identical with cutaneous, lymphatic, and bone tuberculosis.

*b.* The pyogenic form, in which the tubercle bacillus is absent in the lesions or products of inflammation, but which in its outward clinical manifestations closely resembles the tuberculous form. In this form the essential etiological factors are the *Staphylococcus pyogenes aureus* and *albus* and the *Streptococcus pyogenes*.

*c.* The mixed form, in which both the tubercle bacillus and the pyogenic bacteria are found in the lesions and products of inflammation.

**Etiology.**—In considering the etiology of scrofulosis, it should be borne in mind that at the period of life during which the disease occurs the lymph-nodes are not structurally fully developed. On account of this condition and of deficiencies of other tissues such as the skin and mucous membranes, bacteria obtain easy access through the skin, mucous membranes, and lymph-vessels even when there is no breach of continuity of surface (Cornet).

It is also true that certain individuals, once infected, show an inherited predisposition to affections of the mucous membranes and other tissues. This does not mean that scrofulosis as such is hereditary, but that in these subjects exposure to the essential causes of the disease will result in permanently establishing the conditions.

The essential causes of scrofulosis are the tubercle bacillus and the pyogenic bacteria just mentioned. These bacteria are present in ill-ventilated rooms occupied by phthisical patients. Scrofulous infection



may be traced to parents, brothers, sisters, nurses, and playmates. Dried sputum is a prolific source of infection. Infection is favored by any solution of continuity of the skin or mucous membranes, and also by hyperæmia or œdema of these tissues.

The predisposing factors are social conditions, unhygienic surroundings, moist dark dwellings, uncleanness, improper or insufficient food, and lack of fresh air and exercise. The overcrowding in the poorer quarters of cities affords abundant opportunities for infection. Any weakening of the system by infectious diseases, such as measles, pertussis, scarlet fever, diphtheria, rachitis, struma, cretinism, and erysipelas, may be the starting-point for infection. Traumatism or frostbite favors the entrance of bacteria.

**Morbid Anatomy.**—The gross pathological changes are the same in both the pyogenic and tuberculous forms of scrofulosis.

**The Pyogenic Form.**—The mucous membranes are the seat of hyperæmia and thickening. There are increased secretion and activity of the glands, also desquamation of epithelium, and excretion of serum and blood-elements from the surface of the membrane. Adenoids, enlarged tonsils, bronchitis, intestinal and vaginal catarrh, are the most common of the lesions of the mucous membrane.

**SKIN.**—There are eczema, thickening of the epidermis, and transudation of serum and elements of the blood (erythrocytes and leucocytes).

The CORNEA shows conjunctivitis and phlyctenulæ.

The LYMPH-NODES show hyperplasia, which is scarcely noticeable in the early stages. They subsequently enlarge to form tumor masses, which may soften as a result of suppuration or may retrograde to the normal.

**The Tuberculous Form.**—**SKIN.**—Lupus is the form of change found in the skin.

**LYMPH-NODES.**—The nodes in almost any part of the body may be involved. They are enlarged to a greater or less degree, and are infiltrated with tubercle. On section they show either simple caseation or mixed infection. The latter is the case if pyogenic infection is combined with the tuberculous form. Nodes which are the seat of cheesy degeneration may soften and break down, forming cold abscesses. These may open externally or into the bronchi, blood-vessels, pericardium, or peritoneum.

**JOINTS AND BONES.**—In the bones the tuberculous invasion gives rise to fungus or dry caries. Several such foci may be present in the same bone. These foci may heal and years afterward become inflamed as a result of traumatism or infectious disease.

The fingers, toes, and extremities of the long bones are thickened as the result of periosteal inflammation. The ends of the bones are the seat of tuberculous osteomyelitis. The joints may be involved. At first there is serous exudate without perforation into the joint

of the tuberculous foci. Later there are thickening of the synovial membranes and seropurulent exudate into the joint-cavity, with destruction of the cartilages and heads of the bones.

**Symptoms.—General Clinical Picture.**—The patient is anæmic, but not necessarily emaciated; on the contrary, there is a very good panniculus of fat in the majority of cases. The face of some of these subjects presents an eczematous or lupoid eruption. The lips are thick; the conjunctivæ may be injected, and there may be blepharitis or phlyctenula of the cornea. Snuffles and nasal catarrh or ozæna are present. The majority of the patients are mouth-breathers, and suffer from adenoids and enlarged tonsils. In some there is chronic otitis with an offensive discharge. There is a fulness about the neck due to enlarged lymph-nodes. The body may present skin eruptions in the form of ecthyma or varieties of eczema. The general surface is in other cases free from eruption, is pale, and has a transparent, marble-like appearance, showing the blue veins underneath. Many of these patients give a history of chronic bronchitis. In others the remains of old suppurations of the lymph-nodes about the neck are seen in the form of livid cicatrices. If the long bones of the extremities have been affected, the surface of the skin shows either old or recent bone sinuses. The symptoms in most cases develop first on the skin and mucous membranes; the lymph-nodes then enlarge, the bones and joints are next involved, and finally, if the case does not progress favorably, amyloid degeneration of the different organs and emaciation develop as a result of prolonged suppuration. In all cases the changes in the lymph-nodes play a leading part, and are characteristic.

**THE SKIN.**—In the tuberculous variety lupus is the most common form of skin lesion; in another form there is the so-called scrofuloderma of Besnier. Lichen scrofulosorum, with the characteristic enlargement of the lymph-nodes, is another form of skin eruption. In the pyogenic variety eczematous and acneform eruptions are present. In such cases the skin is thickened as a result of chronic inflammations. There are suppurating rhagades around the eyes, mouth, and anus, and ecthymatous eruptions may be present on the trunk and extremities. A form of scrofulous ecthyma, made up of purple painful nodules resembling erythema nodosum, has been described by Hutchinson. Hebra has described a prurigo of the scrofulous subject.

**MUCOUS MEMBRANES.**—There are ulcerations and chronic catarrh of the nasal and bronchial mucous membranes, and in some cases ozæna of an atrophic character. These patients have adenoids and enlarged tonsils. The tonsils are favorite seats of infection. In other cases the posterior nasal and pharyngeal catarrh leads to retropharyngeal abscess, or caries of the spine may cause abscess formations in the retropharynx.



**THE EARS.**—As a result of the catarrh of the nasopharynx chronic otitis may develop. When otitis follows any of the exanthemata in a patient with scrofulous tendencies, it pursues a chronic painless course. Such an otitis may tend to tuberculous disease of the mastoid with sinus thrombosis, or even to tuberculous meningitis. There is pain only when there is a mixed pyogenic infection.

**THE EYE.**—Chronic eczema of the lids, blepharitis, phlyctenula of the cornea, and keratitis fasciculosa are seen. The phlyctenulae do not yield readily to treatment. Hypopyon of the anterior chamber may also be present. Trachoma is in some instances of a tuberculous origin. Lupus of the conjunctiva is sometimes present.

**LYMPH-NODES.**—The tuberculous and pyogenic forms of enlargement of the lymph-nodes are at the outset similar. The pyogenic varieties are associated with enlarged tonsils and adenoids. The skin over the enlarged nodes may remain normal for months or years, or in both the tuberculous and pyogenic varieties it may become adherent, red, inflamed, and break down. The lymph-nodes discharge, leaving suppurating cicatricial openings.

Clinically, infections of the scalp lead to enlargement of the lymph-nodes of the neck and retromaxillary region. Those of the cornea, iris, and ear tend to enlarged preauricular nodes and to enlarged nodes of the submaxillary region. Infections of the mouth and tonsil cause enlarged nodes at the angle of the jaw and beneath it. Otitis with mastoid disease causes enlargement of the node on the point of the mastoid. The lymphatics of the gums and lips are connected with the nodes of the submaxillary region and angle of the jaw. Affections of the nose will cause enlargement of the glands of the neck (Jacobi). Lesions of the fingers will result in enlargement of the cubital and axillary nodes. Infection of a circumcision wound or balanitis will cause enlargement of the inguinal lymph-nodes, as will also infections of the foot and knee.

The lymph-nodes in direct line are always involved; distant ones are never infected unless there is infection of the intermediate nodes. It was formerly believed that the bronchial nodes were particularly subject to infection. Any special susceptibility to infection shown by these nodes is due to their location, infectious material being frequently present in their vicinity.

Cornet found the bronchial nodes affected in 103 out of 126 cases of tuberculous disease occurring before the completion of the fifteenth year. These observations confirm the statement of Henoch, that the bronchial nodes are affected in the majority of cases of tuberculous disease. There are no statistics showing the involvement of lymph-nodes in the pyogenic forms of scrofulosis. Becker, Barthez and Rilliet, Henoch, and Northrup have described the enlargement of bronchial nodes. According to Henoch, they may, even if tuberculous, be enlarged without involving the lung tissue.

By pressing on the vagi they may cause rapidity of pulse, and if on the recurrent laryngeal may give rise to spasmodic dyspnoea or to a croupy cough. Pressure on the œsophagus may cause dysphagia; pressure on the trachea may cause inspiratory dyspnoea; and pressure on the pulmonary veins, hyperæmia of the lungs. Hensch and Baginsky doubt the possibility of diagnosing these enlarged nodes even with the help of all these symptoms.

These nodes may retrograde to the normal size (West) or they may break down and perforate into a bronchus or the trachea. If they perforate into the pericardium, pleura, or mediastinum, inflammation results at these points.

The mesenteric lymph-nodes may enlarge and cause pain or tuberculous infection of the peritoneum (*tabes mesenterica*). In some cases they may be palpated through the abdominal wall.

**THE BONES AND JOINTS.**—The extremities of the long bones are most frequently the seat of disease; the diaphysis rarely so. The phalanges of the fingers, the toes, the radius, the ulna, and fibula, are affected in the order of naming. The joint-cavities may at first contain exudate without perforation of the cartilage; later, pus is found in the cavity.

All of the structures of the joint are involved, and the joint may eventually be destroyed. Suppuration of a chronic nature may, as stated elsewhere, tend to amyloid degeneration of the liver and spleen.

There is, in addition, a progressive anæmia. The temperature is sometimes raised a half or three-quarters of a degree above the normal, at others it is normal. Exhausting sweats occur; the disturbances of nutrition become in some cases extreme. There may be intestinal diarrhoea.

**Course and Prognosis.**—This condition is not necessarily fatal. Many cases make a good recovery under proper management. This is particularly true of the pyogenic form. The tuberculous variety may also retrograde if the disease be localized to certain lymph-nodes or bone foci.

**Diagnosis.**—The diagnosis of the pyogenic form is made from the clinical history; that of the tuberculous variety, either from the presence of the tubercle bacillus in the pus or lesions of the disease. There are mixed forms in which it is not always possible to decide whether the process is tuberculous or pyogenic. The clinical history and blood examination will be of service in differentiating scrofulosis from leukæmia, pseudoleukæmia, and lymphomata of a malignant nature (Plate XII.). In other cases the diagnosis from late forms of hereditary syphilis cannot always be readily made. The history of such cases is of importance. In many cases a resort to antisyphilitic treatment will be necessary to complete the diagnosis.



PLATE XII.



Multiple Lymphosarcomata as Differentiated from Scrofulous Lymphadenitis. Enlargement of the cervical, supraclavicular and axillary lymph-nodes. Child six years of age.





The **treatment** of scrofulosis is directed toward limiting if possible the spread of the infection, preventing reinfection of the patient, and instituting local treatment of the lesion. In order that the disease may be treated successfully, the patient should be placed under hygienic surroundings. If the patient is in the city, removal to the country is advisable. The food should be plain and nutritious; milk, eggs, meat, vegetables, and cereals should form the diet. The hygiene of the skin is important. Alkaline or sea baths give tone to the skin. Moderate exercise in the open air is also of great service in correcting the anæmia and tendency to inaction shown by these patients. In a word, the patient should be removed from the conditions and surroundings which originally induced the infection.

The medical treatment is limited to the exhibition of such tonics as iron, Fowler's solution, and strychnine. The intestines should receive attention during the administration of iron. Fowler's solution gives better results in pyogenic lymphadenitis than in the tuberculous form. The syrup of ferric iodide in full doses has a tonic effect on the mucous membranes. Baginsky advises the exhibition of preparations of thyroid gland. I have not seen any markedly good results obtained by this method of treatment.

Cod-liver oil is of great value in this disease. In the form of emulsions it should be given in full doses; with young children its use must sometimes be suspended on account of the laxative effect on the intestines.

The local skin lesions should receive appropriate treatment, as should also the bones, joints, and suppurating lymph-nodes. It is not within the province of this work to enter upon the surgical details of such treatment.

### TUBERCULOSIS.

**Etiology.**—The essential cause of tuberculosis is the tubercle bacillus. This micro-organism gains entrance to the body through the respiratory channels (inhalations), the intestinal canal, and through wounds. The air in ill-ventilated rooms occupied by tuberculous individuals may at times contain the bacillus in particles of dried sputum. Any abrasion of the mucous membrane of the respiratory tract affords opportunities for the entrance of the bacillus into the blood and lymph-channels. Among cases of this kind belong those in which phthisical individuals have breathed into the mouths of asphyxiated infants or children.

Not much importance is attached by certain authors to the possibility of infection through the intestinal canal (Baumgarten, Bollinger). However, cases have been reported in which infected cows' milk has caused tuberculosis in infants (Leonhardt, Sonntag, Eber). Infection in this manner is rare, because the food of infants (milk) is as a rule heated before it is taken into the stomach.

In a recent monograph Blackader says that of 125 autopsies on tuberculous children Northrup found that in 88 cases the primary infection had occurred through the respiratory tract. In 3 cases the pathway of infection was the intestinal canal, and in 35 cases the primary seat of infection remained undetermined. Of 75 additional autopsies, Bovaird found the primary lesion in the lungs or bronchial nodes in 60 cases. English writers, according to Blackader, place the frequency of primary infection through the intestinal tract at a much higher figure (25 to 30 per cent.) than do American investigators.

Still, of Great Ormond Street, gives the following statistics of primary tuberculosis in infants and children :

Lung . . . . .	105	} 138	} 153=57 per cent.
Probably lung . . . . .	33		
Ear . . . . .	9	} 15	
Probably ear . . . . .	6		
Intestine . . . . .	53	} 63=23.4 per cent.	} 53=nearly 20 per cent.
Probably intestine . . . . .	10		
Bones, etc . . . . .	5	} 53	
Fauces . . . . .	2		
Uncertain . . . . .	46		

German pathologists maintain that primary tuberculosis of the intestinal canal is rare. Marfan, the French pediatricist, places the frequency of primary intestinal tuberculosis at 87 per cent.

In general, it may be said that the avenue of infection in infants and children is through the respiratory tract. Infection through infected milk is now believed to be very rare. The importance of this mode of infection has been heretofore much exaggerated (Blackader).

Infection through mother's milk has been recorded as occurring in the newborn infant (Roger and Garnier). Heredity only creates predisposition to the disease, as it does with adults. Dennig found that 58 per cent. of his cases of tuberculosis occurred in children of tuberculous families.

Infection through wounds is very uncommon with children, because they are not usually exposed to traumatism. It is not so likely to result in general tuberculosis as infection through the other channels. This is proved by the fact that in the adult so-called autopsy tubercle rarely tends to general infection. Infants who are subjected to ritual circumcision are exposed to infection through the practice of arresting the resulting hemorrhage by mouth suction. I have seen eight instances of such infection. In these cases there is a primary enlargement of the inguinal lymph-nodes before the process becomes general. The vertebræ may become tuberculous or meningitis of a tuberculous nature may result.

Tuberculosis may be conveyed to the fetus either through the placenta, through the spermatozoön, or the ovum of the mother.



Under placental infection are to be included those cases in which the tubercle bacillus has been found in the blood of the fœtus without accompanying changes in the organs (Schmorl), and those in which tubercle nodules and enlarged lymph-nodes have been found at birth (Landouzy and Lehman). In both these forms of tuberculous infection the mother had suffered from acute miliary tuberculosis. The spermatozoön and testis may contain tubercle bacilli in the absence of gross tuberculous lesions of the organ (Nakarai and Kockel). Tuberculosis may in this way be conveyed into the uterus at the time of conception. Jahni and Weigert found tubercle bacilli also in the Fallopian tubes of women dying of phthisis, although there were no gross changes in the tubes. The ovum may thus convey tubercle bacilli. As a rule the bacilli thus introduced into the ovum of the fœtus are dormant during intra-uterine life. They may develop at any period after birth (Baumgarten). Additional facts supporting the theory of intra-uterine infection of the fœtus have been reported by Baumgarten and Roloff, who found a cheesy nodule in a dead-born fœtus, and by Birch-Hirschfeld and Bugge, who found bacilli in the blood of a fœtus born of a tuberculous parent. In 45 per cent. of the infants born in the Bohemian Foundling Asylum, tuberculous lesions were present to such a degree that the congenital nature of the affection was unmistakable (Houl).

The infectious diseases play an important rôle as predisposing factors in tuberculosis. Measles, scarlet fever, pertussis, and influenza, by lessening the resistance of the economy and impairing the integrity of the air-passages, favor the infection. Tuberculous bronchopneumonia occurs under these conditions, either because the tubercle bacillus was present in the body before the infection was contracted or gained access subsequently (Fränkel). In the majority of cases the former condition is the rule. Cold, unhygienic surroundings, and poor food, all predispose to infection as with adults.

The frequency of the occurrence of tuberculosis in infancy and childhood varies with the environment. Statistics are therefore only relative. Seidl found that of 646 consecutive autopsies upon children, 14 per cent. were tuberculous. Forty-five per cent. of all the cases of tuberculosis occur during the first two years of life, 25 per cent. of the total number occur during the first year, and 71 per cent. during the first five years (Queyrat, Lannelongue, Dennig). It is slightly more frequent among girls than boys.

### **Pulmonary Tuberculosis.**

Seventy per cent. of the infants and children who die from tuberculosis show lung-changes (Dennig). Infection first occurs through the respiratory tract. A cheesy lymph-node may burst into the bronchi, and bacilli may thus gain access to the lung alveoli and

cause changes, as they do in the adult lung. Hæmatogenous infection occurs through the bursting of a small tuberculous nodule into a bloodvessel, thus flooding the lung with infectious matter, or by the carrying of minute emboli of this material to distant parts of the lung.

Tuberculous bronchial lymph-nodes, bone, and pleura may also give rise to infection of the lung through the lymph-channels. The part played by the infectious diseases in its dissemination has been already mentioned.

**Morbid Anatomy.**—The three principal forms of tuberculosis of the lungs which occur in infants and children are :

The **miliary form**, which is characterized by the eruption of miliary tubercles throughout the lung. The lung is on section found to be dark red, hyperæmic, and to contain less air than the normal lung. The bronchial mucous membrane is hyperæmic and covered with blood and mucus.

The **cheesy or cheesy ulcerative form**, also called florid phthisis, takes the form of cheesy lobar or lobular pneumonia. In recent cases the lung is grayish red, and there are areas which rapidly become cheesy, and are not encapsulated. These may coalesce, involving the greater part of a lobe in the process. Small cavities are frequent, large ones rare. The cheesy ulcerative form occurs as a result of the aspiration of large numbers of tubercle bacilli.

The **chronic form**, which is a cheesy fibrous bronchopneumonia, is essentially a tuberculous bronchopneumonia. Round cheesy nodules are found surrounded by a fibrocellular zone resulting from the destruction of extensive areas of lung-tissue. The pulmonary pleura is thickened. The bloodvessels participate in the process. There is endarteritis with miliary tubercle in the walls of the bloodvessels, and there may be thrombosis. The tubercles may burst into the interior of the bloodvessels. The bronchi, trachea, and larynx may be affected. There are ulcerations of the mucous membrane and destruction of cartilage. The bronchial lymph-nodes or glands are enlarged and infected in most cases of tuberculosis of the lungs in children. Henoch has, however, shown that the bronchial nodes may be tuberculous and greatly enlarged without involvement of the lung-tissues. Northrup found the bronchial lymph-nodes affected in 125 consecutive autopsies. The whole node is converted into a cheesy mass, which may soften and break down. If there is a perforation into a bronchus, masses of bacilli may be discharged into the lung. Perforation into the bloodvessels may also occur. The nodes may form small masses or large mediastinal tumors at the root of the lung.

**Localization.**—The apices of the lungs of infants and children are not as in adults the region most frequently affected by tuberculosis. The first change may appear in the lower lobe or the lower portion



of the upper lobe, and spread thence. This is accounted for by the miliary character of the affection in the lungs of infants and children (*Rindfleisch*), and also by the fact that in many cases the process spreads from the bronchial nodes or glands to adjacent parts (*Weigert*).

The **symptoms** of tuberculosis of the lungs in infants and young children are not so characteristic as in the adult, nor is there a gradual development of the symptoms pointing to involvement of the lungs. After the fifth year of life the symptoms closely resemble those seen in the adult. As regards infants, we shall describe only clinical types of the disease. Even these exhibit many varieties.

*Henoch* has described forms of tuberculosis in infants which closely resemble cases of *marasmus* due to gastro-enteric disease. In many of them there are steady emaciation and progressive muscular weakness; the infant lies helpless; the abdomen is retracted; the eyes may present a conjunctivitis; the cervical, axillary, and inguinal glands may be slightly enlarged; there is constipation alternating with diarrhoea; the skin is easily inflamed and abscesses may form. In the terminal period vomiting sets in. The lungs throughout the course of the disease may present few signs, or there may be evidences of a general bronchitis. In these slowly emaciating infants there is no cough of sufficient severity to indicate involvement of the lung. The terminal stage may present cerebral symptoms of a mild type, such as rigidity of the neck, with periods of stupidity alternating with irritability. The infants die with a progressive loss of flesh and strength. The temperature is for days normal or a little above normal. In other types the disease is masked by an acute or subacute bronchopneumonia. In these cases the infant, after suffering from exposure or some infectious disease, suddenly exhibits all the signs of a bronchopneumonia. There are severe cough, high temperature, dyspnoea, and cyanosis, as in the ordinary bronchopneumonia. Death may ensue in a few days or in a week. In other forms fatal results take place after several weeks, with symptoms closely resembling those of a persistent bronchopneumonia of the ordinary non-tuberculous variety. In other cases the symptoms of an acute bronchopneumonia are present, sometimes complicated with empyema. Evacuation of the pus is followed by apparent improvement, and the empyema may even heal, but the infant or child gradually emaciates, and the cough, which may have abated, becomes aggravated. Examination of the chest reveals new areas of lung involvement. In these cases the pus does not always contain the tubercle bacilli. The empyema may be the result of mixed infection, and the pus may contain only simple streptococci, the physician being frequently misled as to the true condition. Many forms of tuberculosis of the lungs in infants and children

cause death with the terminal symptoms of tuberculous meningitis. Especially characteristic in older children, as compared with the adult, are those cases of tuberculosis of the lung which follow some slight injury, blow, or exposure, and in which there are for weeks no signs in the lung or elsewhere to account for the gradual emaciation and intermittent or remittent temperature. After a variable length of time signs of involvement are detected at one apex, or posteriorly over the base or mid-area of the lung. Even then the cough may be absent and no sputum be expectorated. The child then has intervals of stupidity; there is delirium at night accompanied by the typical hydrocephalic cry. Irritability of temper is marked, the emaciation is very rapid, and coma and death with terminal paralyses show that the infection has involved the cerebral meninges.

The TEMPERATURE is irregular in course. It may be normal for a few days, after which it rises one or two degrees daily in the afternoon and falls to the normal toward morning.

HÆMOPTYSIS is very rare in infants. Henoch has seen 3 cases in young infants and 1 in a child of two years. Acker has reported a case in a child of three years. I have seen several cases in children of more than six years of age.

SPUTUM.—Infants do not expectorate. At most a frothy mucus collects around the orifice of the mouth after a coughing spell. Even older children expectorate very little, and must be taught to do so.

**Course.**—Up to the second year of life, the course of tuberculosis of the lung is generally acute (Henoch). The disease may pursue a subacute course, but it is rarely as prolonged as in the adult. In children above the fifth year its course closely resembles that taken in the adult.

The **diagnosis** of tuberculosis of the lung in infancy and early childhood must, for the most part, be made from the history of the case. In many of the cases the physical signs in no way differ from those seen in non-tuberculous diseases. Cases in which marked consolidation of the lung persists, with progressive emaciation, and cases in which auscultation reveals the presence of cavities, are certainly suspicious. There is no reliable method of determining the nature of an acutely developing bronchopneumonia; the detection of the tubercle bacillus in the vomit, in the feces, or in the exudate of a complicating pleurisy or empyema, is of diagnostic aid. The use of tuberculin as a diagnostic agent has not met with the approval of the profession.

The existence of enlarged lymph-nodes in the mediastinum or the root of the lung is, according to some authors, revealed by symptoms of pressure. Pressure on the bronchi may give rise to dyspnoea; on the large veins, to nervous congestion and cyanosis, or



œdema of the lungs; on the recurrent laryngeal nerves, to asthma or laryngospasm; on the œsophagus to dysphagia. Although in exceptional cases such symptoms may be thus correctly interpreted, I believe with Hænoch that diagnosis of these enlarged nodes during life is highly uncertain.

**Treatment.**—From a study of the symptomatology it will be seen that the treatment of tuberculosis of the lung in young infants and children must be simply symptomatic. A case of suspected tuberculosis should be isolated from other children. The fever needs little attention if it remains low; if high, it is treated as in a case of simple bronchopneumonia. The cough and restlessness are also treated symptomatically.

### Tuberculosis of the Peritoneum.

(*Tuberculous Peritonitis.*)

**Occurrence.**—According to the statistics of Dennig, Müller, Biedert, and Simmonds, tuberculous peritonitis occurs in from 8 to 21 per cent. of all the cases of tuberculous disease. Sixty-five per cent. of the cases operated on by Herzfeld were under the age of fifteen years. The frequency varies in different localities. The figures given by different authors vary with the nature of the material utilized for statistical purposes.

**Etiology.**—Tuberculous peritonitis is rarely if ever primary, although such cases have been described by Hænoch and Müller. The peritoneum may become infected through the blood-channels (hæmatogenous); under these conditions tuberculosis of the peritoneum is simply a feature of the manifestation of acute miliary tuberculosis. The peritoneum may become infected through the lymphatics or lymph-channels (lymphogenous). Under these conditions it is the result of infection from adjacent organs, such as the intestines, the genito-urinary tract, the mesenteric, peritoneal, or retroperitoneal lymph-nodes, and the vertebrae and pleura.

**Morbid Anatomy.**—There are, according to Herzfeld, three main forms of tuberculous peritonitis: the miliary, submiliary or exudative form; the nodular or sclerosing form; and the adhesive form.

**The Miliary, Submiliary, and Exudative Form.**—In this form there is an eruption on the peritoneal surface, of gray, transparent tubercles of varying sizes. The intestinal coils are covered with fibrin, and are slightly adherent to one another. There is a clear serous, sero-fibrinous, seropurulent, or even ichorous exudate (mixed infection).

**The Nodular or Sclerosing Form.**—In this form the quantity of the exudate in the abdominal cavity is small. The omentum is converted into a solid cylindrical mass, containing tumors of a tuberculous nature as large as an apple. The mesentery is thickened and

covered with tubercles. The intestinal wall is thickened and covered with gray or grayish-yellow tubercles, which may attain the size of tumors. The coils of gut are adherent, and the whole peritoneal cavity may be obliterated.

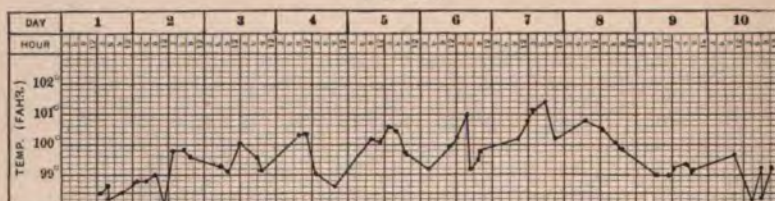
**The Adhesive Form.**—In this form the intestines form an adherent mass, with masses of exudate between the coils of gut, forming pseudocysts. This exudate may be of a puriform nature. Aggregations of tubercles between the coils of gut break down and perforate into the gut, or become adherent to the abdominal wall and perforate externally forming intestinal or abdominal fistulæ. Perforation may thus occur in the absence of any real ulceration on the mucous membrane of the gut.

In addition to the above principal forms of tuberculous peritonitis, mixed forms occur.

The exudate in the peritoneal cavity may be purely serous (ascites), or the serum may, as in a case which I observed, have a chylous appearance, due to the admixture of fat. In other forms the exudate may be seropurulent, hemorrhagic, or, in mixed infections, putrid. In the purely ascitic variety the fluid is free; in the purulent form, it is frequently sacculated between the adhesions on the coils of gut.

**Symptoms.**—The disease is, as a rule, insidious and slow in development. The stage of abdominal distention has usually been

FIG. 67.



Tuberculous peritonitis. Female child, five years of age. Ten days of her temperature immediately preceding operation (laparotomy).

reached when the patient is first brought to the physician. The history shows that the child has been for some time gradually losing weight, that the appetite is capricious, and that there have been attacks of abdominal pain. This pain may be localized or radiate from one point, may be constant, or may resemble visceral neuralgia. Sometimes there is no history of pain, but it may be detected by pressure on parts of the abdomen. There may be a slight rise of temperature toward evening (Fig. 67); diarrhœa may alternate with constipation. The abdominal distention is the leading feature. It may take the form of a uniform ascitic accumulation (Fig. 68); the surface of the abdomen may be uneven and irregular, and tumors with cystic formation may be felt through the abdominal walls.



The movements, which are rich in fat, sometimes resemble icteric evacuations. This condition was formerly considered pathognomonic of tuberculous peritonitis (Biedert, Conitzer).

Vomiting of fecal or biliary matter resembling that seen in appendicitis may occur.

In marked contrast with these is a form which in its acute onset may simulate acute perforative peritonitis. In this variety the tubercle mass may cause perforation either of the appendix or the gut in its cavity. Symptoms of acute perforative peritonitis which in every way resemble those of appendicitis set in. It is only by resort to laparotomy that the nature of the affection can be discovered.

**Physical Signs.**—The physical signs in the miliary and the nodular forms are due to the presence of free fluid in the abdominal cavity. If ascites is present, there will be the percussion-wave, the flatness in the flanks, and change of the tympanitic area will occur with change in the position of the patient. If adhesions are present and there are encapsulations of fluid, the signs will not vary on changing the position of the patient. On the other hand, in the adhesive form there will be evidences of tumor masses in the abdominal cavity, cystic formations caused by the encapsulated exudate, and little or no fluid.

In cases of adhesions in tuberculous peritonitis of the miliary form, the fact that when the patient is in the recumbent position the coils of gut may here and there be seen outlined over the abdominal parietes, is of diagnostic value (Fig. 69). I was able by this means to confirm the diagnosis of adhesions in one such case, and have detected them clinically in other cases in which this form of peritonitis had been diagnosed.

The liver may be enlarged as a result of amyloid degeneration or tuberculous interstitial hepatitis.

The spleen may be enlarged as a result of amyloid degeneration.

Rectal examination may reveal miliary nodules or peritoneal masses palpable through the walls of the rectum.

FIG. 68.



Uniform abdominal distention due to ascites of tuberculous peritonitis; enlarged spleen.

The **diagnosis** is based on the slow and insidious onset, the colicky abdominal pains, abdominal tenderness on palpation, the presence of ascites or tumor masses, constipation alternating with diarrhœa, progressive loss of strength, intermittent fever or slight rise of temperature in the evenings, and the presence of tuberculosis in other organs. At the outset tuberculous infection in other parts of the body may be difficult of detection. A rectal examination should always be made. This form of peritonitis should be differentiated from the non-tuberculous form. Inasmuch as some authors, notably Unger and Nothnagel, doubt the occurrence of

FIG. 69.



Tuberculous peritonitis, miliary form, female child, five years of age. Irregular contour of abdominal parietes in the recumbent posture, showing intestinal agglutination.

idiopathic non-tuberculous peritonitis, caution should be exercised in making a diagnosis of simple chronic peritonitis. Absence of emaciation and retrogression of symptoms by no means prove that the



disease may not have been tuberculous, since some forms of tuberculosis of the peritoneum present such peculiarities.

This form of peritonitis must also be differentiated from cirrhosis of the liver, new growths, cardiac and renal affections.

**Course.**—The course of the disease is chronic. Frequently the symptoms retrograde and there is an apparent recovery. The ascites may at times diminish, and again increase. The chronic forms unless operated upon lead to the formation of abdominal fistulæ, to perforative peritonitis, to tuberculosis of the organs, and to amyloid degeneration of the liver and spleen, with emaciation, exhaustion, and death.

**Treatment.**—Laparotomy, when advanced tuberculosis in other organs is not present, is, according to Herzfeld, curative in 54 per cent. of cases. In a series of 29 cases of all ages operated upon by him, 19 were under the age of fifteen years. With operative treatment must also be combined the medicinal and hygienic treatment suitable to cases of pulmonary or local tuberculosis. On the other hand, in the forms which resemble cases of *tabes meseraica*, in which emaciation and cachexia are present before much exudate is formed, it is difficult to decide as to the propriety of operative measures, especially if diarrhœa be present. In these cases if the diagnosis is not certain, proper feeding should be begun and the condition of the patient improved before laparotomy is attempted.

#### Simple Chronic Peritonitis.

Although Henoch and Müller have reported cases of chronic idiopathic non-tuberculous peritonitis, its occurrence is still a matter of dispute. Nothnagel, Unger, and Heubner, while not denying *in toto* its possible occurrence, insist on its extreme rarity. The absence in these cases of progressive emaciation is no proof of the non-tuberculous nature of the affection. The absence of the tubercle bacillus in the abdominal exudate is of slight diagnostic value. In 29 cases of undoubted tuberculosis of the peritoneum Herzfeld found the bacillus only once in the ascitic fluid. In some forms of tuberculous peritonitis the nutrition may not only be good, but there may be no history of heredity or scrofulosis. It is manifest that under these conditions it is impossible to describe a disease the existence of which is still in doubt.

#### Other Forms of Tuberculosis.

**Tuberculosis of the larynx** is rare in children. It occurs in from 3 to 4 per cent. of the total number of cases of tuberculosis (Reiner, Steffen, Barthez, Rilliet). Demme has reported a case in a child of four and one-half years.

**Tuberculosis of the Pleura and Pericardium.**—Primary

tuberculosis of the pleura is rare. Dennig reports that it occurred as a feature of general tuberculosis in 14 per cent. of his cases. Pericarditis of the tuberculous variety occurs in only 3 per cent. of the cases of general tuberculosis.

Tuberculosis of the heart muscle is very uncommon. Säger reports a case in a child of nine months, and Demme one in a patient of five years. The endocardium may be involved in general tuberculosis (Perroud).

### Abdominal Tuberculosis.

The following table, showing the relative frequency of tuberculous involvement of the abdominal viscera, is taken from Dennig's figures :

	Peritoneum.	Stomach and intestines.	Mesenteric lymph-nodes.
Dennig . . . . .	8 per cent.	14 per cent.	21 per cent.
Müller . . . . .	18 "	38 "	57 "
Biedert . . . . .	18 "	31 "	40 "
Simmonds . . . . .	21 "	31 "	53 "

### Tuberculous Meningitis.

(*Acute Internal Hydrocephalus ; Basilar Meningitis.*)

**Occurrence.**—Tuberculous meningitis has been observed in infants as early as the third month (Steffen). Barthez and Rilliet have seen cases in infants five months old. The frequency of tuberculous meningitis varies with the locality. Dennig places the frequency of tuberculous meningitis among children who suffer from tuberculous disease at 60 per cent., while Medin found this form of meningitis in 15 per cent. of tuberculous children. It is most frequent in the nursing period ; 75 per cent. of all cases occur under the fifth year. The second year of infancy shows the greatest number of cases (Steffen). It is more frequent among male than female children.

**Etiology and Morbid Anatomy.**—Exposure to cold and traumatism predispose to the affection. In many cases there is, in addition to the meningeal disease, disseminated tuberculosis of the lungs, pleura, spleen, liver, and peritoneum. In other cases the meninges are the chief seat of the disease, only a few isolated foci of tuberculosis being present elsewhere, as in the mesenteric or bronchial lymph-nodes. It is rare to find the lesions confined to the meninges, and some authors deny the possibility of such a condition. It is not always possible to determine the primary focus of infection.

The tubercle bacilli, which are the causative factors, may be carried by the blood (hæmatogen) to the meninges, and there give rise to a more or less extensive miliary deposit. The original focus



PLATE XIII.



Tuberculous Meningitis. Infant eight months of age. Stage of paralysis, left facial paralysis; left lagophthalmus, bulging fontanelle.





in such cases may have been a cheesy lymph-node, a tuberculous nodule in the lung, or a carious bone or joint. The tubercle bacilli may enter the lymph-channels from an adjacent carious bone of the skull, a diseased mastoid, or a spinal vertebra. An ozæna or a solitary tubercle of the brain may give rise to tuberculous meningitis by this mode of transmission. Whatever the focus, at the point of infection there will result an eruption of tubercles which may remain localized or become disseminated along the course of the cerebral lymph-channels. When the dissemination of bacilli occurs through the blood, miliary tubercles are found in the course of the bloodvessels and in their walls. The tubercles may remain confined to the meninges or may involve the brain-tissue, constituting a meningo-encephalitis. In the meshes of the meninges (pia) there is a serofibrinous or seropurulent exudate, which is also found in the ventricles (hydrocephalus), and which infiltrates the brain-tissue itself (meningo-encephalitis). If the exudate is limited in quantity and the tuberculous process is localized, forming one, two, or three nodules, we speak of tuberculosis of the brain or solitary tubercles of the brain. The tubercles are seen as grayish shining nodules surrounded by a hyperæmic zone. They vary from microscopic size to large yellow nodules, which are of older origin. They may be strung along the vessels or break through their walls, forming thrombi with subsequent hemorrhagic infarction.

The basal branches of the artery of the Sylvian fissure are usually the seat of the eruption of these tubercles. The tuberculous process is generally bilateral, but one side, usually the left, may alone be the seat of disease. Tuberculous meningitis affects by selection the base of the brain—hence the term basilar meningitis. The convexity may be involved in the process, although it is rarely the seat of tuberculous meningitis unless the base of the brain is affected. Steffen and Henoch have described cases of isolated tuberculous meningitis of the convexity of one or both sides. The base, the vicinity of the pons, and the chiasm are the seats of the process. In all forms, the pia is infiltrated with serofibrinous or seropurulent exudate. The choroid plexus may be involved. Acute hydrocephalus then results.

The brain-tissue itself is the seat of inflammatory infiltration, and the nerves at the base are surrounded with exudate and are in process of degeneration. The ependyma of the ventricles (the lateral, third and fourth) are the seat of inflammatory thickening and eruption of tubercles. These cavities are filled with exudate. The meninges of the spinal cord, the cord itself, and the nerves at the points of exit are frequently involved in the tuberculous meningitis, as has been demonstrated by Leyden, Erb, and Dennig. The latter found the cord involved in 9 out of 10 cases. The pia may be involved to a slight degree. The nerves at the points of exit are

involved in inflammatory exudate. The tissue of the cord and the nerve-elements may be the seat of degenerative processes.

The **symptoms** of tuberculous meningitis cannot be clearly classified according to stages. There is an indefinite period of premonitory symptoms followed rather abruptly by manifestations of cerebral irritation, and ending with a period in which pressure-symptoms are pronounced. As a rule, the disease is slow of development, although cases occur in which the rapid malignant course simulates that seen in rapidly fatal cerebrospinal meningitis of the epidemic type. The disease gives a varying clinical picture in the different periods of childhood. The infant of from seven to twelve months refuses to nurse, has a low fever, and may have diarrhoea alternating with obstinate constipation. The illness of an infant is often attributed to a fall occurring while it was learning to walk. A weakness of the extremities is thus indicated. The infant becomes indifferent to its surroundings and passes into a somnolent condition. Emaciation is progressive. Vomiting occurs once or twice daily, the food being ejected from the mouth after nursing without apparent effort. The vomiting may be followed by a convulsion, after which the infant becomes unconscious. There may be strabismus, or rigidity of the extremities, or the extremities may be in constant motion of an automatic character. The convulsions may follow one another without cessation. These symptoms may set in after a period of one, two, or five weeks of ailing. In other cases the infant may have suffered from a chronic otorrhoea, although otherwise in apparent health. Suddenly, vomiting followed by a convulsion sets in. This convulsion is the forerunner of symptoms, such as coma, which denote that the disease has become established without having attracted the notice of the parents. In children of five years of age the symptoms are more marked. The child may have an attack of vomiting and diarrhoea and apparently recover; after a few weeks, during which there are irritability, loss of appetite, and progressive emaciation, the child no longer desires to be up and about, but lies quiet in its crib, with its head in a characteristic rigid position. It develops strabismus, becomes soporose, and cries out at night. This cry is sometimes piercing in character, and is the cause of much concern to the mother. When the symptoms of cerebral pressure are fully developed, the picture is in the majority of cases much the same. The infant after the first convulsion lies in a soporose or comatose condition. The eyes are open and there is a vacant stare; the sclera may be apparent above the cornea; the fontanelle if still open is tense and bulging, and there may be horizontal nystagmus. The infant cries if disturbed, or may be indifferent to its surroundings. The pupils may be unequal in size and react to light. In one case which I observed the pressure-symptoms were extreme. The infant lay on its back with rigid neck and arched back (opisthotonos), and emitted a piercing cry at in-



tervals. At each cry the pupils became successively dilated and contracted (hippus). I have seen this phenomenon in two cases of tuberculous meningitis. Opisthotonos may be present, and the retraction of the head may relax at intervals, the muscles of the back being lax. In some cases there is apparently no rigidity of the neck. As a rule there are no convulsions. As the infant or child lies quietly in its crib the inspirations during the stage of cerebral pressure may be very irregular or may be of the Cheyne-Stokes type. The outline of the abdomen is at first normal or there may be a slight retraction at the upper part. The abdominal wall may be quite lax, so that the coils of gut can be made out. If

FIG. 70.

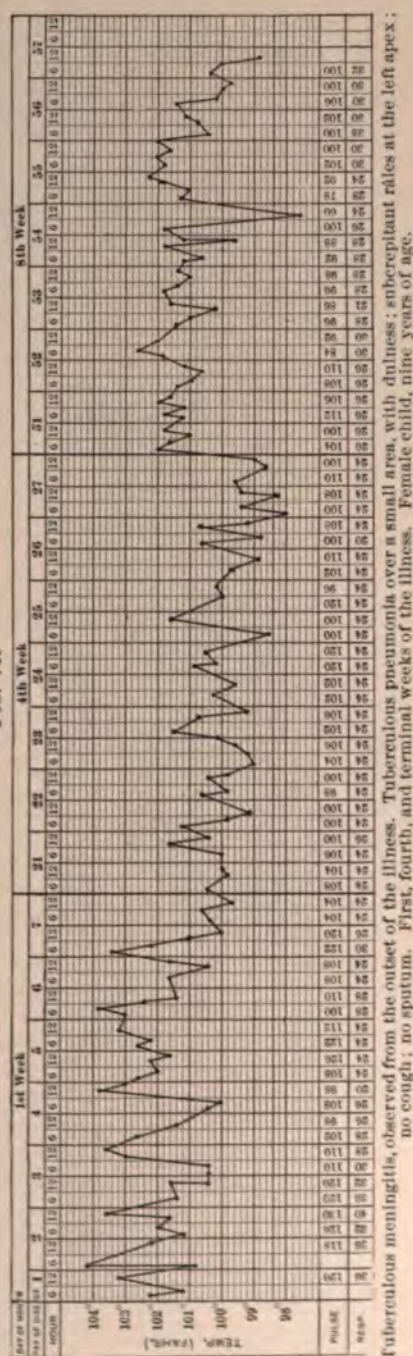


Babinski's reflex. Tuberculous meningitis; stage of facial palsies. Boy, seven years of age.

the case is protracted, retraction of the abdomen occurs in the final stages of the disease. This condition has been described as the boat-like abdomen. It is not diagnostic of this form of meningitis.

In rare cases spastic symptoms closely resembling those of tetany occur after the initial convulsion. The infant lies comatose, with rigidly flexed arms; the Chvostek and Trousseau symptoms are present. In all of these cases, if the skin is stroked with the finger ever so lightly, a red mark appears over the stroked area (*tache cerebrale*). In the spastic cases the knee-reflexes may be increased, but in the non-spastic cases they are diminished. It is difficult to elicit Kernig's symptom in spastic cases, because the infants lie with the

Fig. 71.



Tuberculous meningitis, observed from the outset of the illness. First, fourth, and terminal weeks of the illness. Female child, nine years of age.

knees flexed. By straightening the legs and thighs it is possible in the majority of children to obtain the symptom.

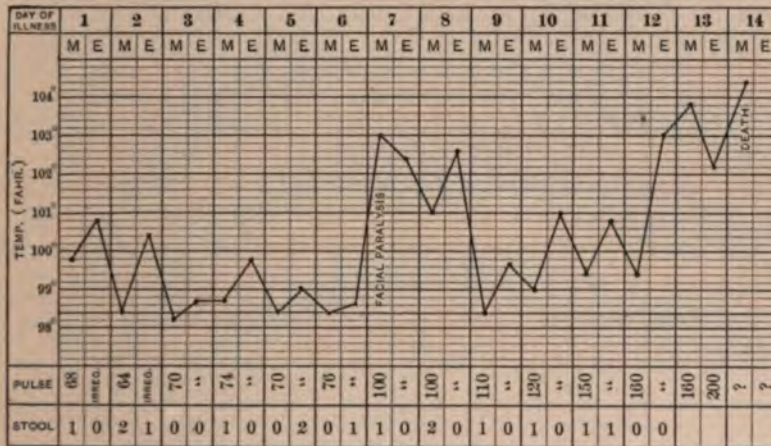
The most important symptoms of the final stage of tuberculous meningitis, both in infants and older children, are the localized facial palsies. For several days or weeks preceding the fatal issue, one side of the face is seen to be flatter than the other. There may be ptosis or lagophthalmus of the eyelids. One eye may be rotated internally, owing to paralysis of the abducens. The extremities are also parietic. The arm and leg of one side may be rigid or flexed, while those of the opposite side are lax. Irritation of the soles of the feet may give a Babinski reaction (Fig. 70). In some cases this reaction is present independently of any irritation of the plantar surface. Toward the end, convulsive twitchings appear in the muscles of one or the other side of the face or of the extremities. Death supervenes in coma with convulsions. The heart may continue to beat for some time after the cessation of respiration.

Children from six to nine years of age present a more decided clinical picture in the premonitory stage. For some weeks before the onset of symptoms of irritation they complain of headaches, frontal, sincipital, or parietal.



The patient is listless, walks with an unsteady gait, and has no desire to study or play. A discharge from the ear may have been present for months before the onset of these symptoms. In one case the child had for some time complained of pain in the left side of the chest and had lost weight steadily. There were mild pleurisy and signs of slight consolidation at the apex of the left lung. There was daily elevation of a few degrees of temperature in the evening, and a normal temperature in the morning. In this case, although there were distinct signs of pulmonary involvement of a mild type, the emaciation was progressive and the leucocyte counts low (8000 W.B.C.). At night the typical cry of tuberculous meningitis was present. In the early stages of the disease the patient was conscious during the day, but later became listless, irritable, and slept or was drowsy during the day.

FIG. 72.



Tuberculous meningitis, observed from the outset of the symptoms. Female infant, fourteen months old.

When questioned, a slow stupid answer was given. The child vomited and at times became nauseated. The Kernig symptom appeared. Right lagophthalmos was present. The pupils were unequal in size, the left being dilated. The pulse at this time varied from 60 to 100 and was compressible. Finally, coma set in with left facial palsy and convulsive twitchings of the left side of the face. This case was for three months under constant observation. In other cases the vomiting is rapidly followed by paralytic symptoms such as ptosis and facial paralysis on the same side. There are no convulsions and no cry, but there is rigidity of the neck and extremities; one patellar reflex may be absent. The Kernig symptom and Babinski reflex are present in the majority of cases in children.





be reached in the morning hours and the lowest toward evening. In the majority of cases, however, the temperature is rarely higher than 103° F. (30.4° C.).

The pulse is increased at the onset, but during the course of the disease becomes slow and may range from 60 to 100 or more during the twenty-four hours.

The respirations are irregular, and may vary from 18 to 60 within the twenty-four hours, even if no pulmonary lesion is present.

The **diagnosis** of tuberculous meningitis is based upon the history, the general symptomatology, and the results of lumbar puncture. A history of tuberculosis in the father or mother or the presence of a tuberculous focus is significant. Chronic otitis, disease of the bones or joints, and signs of lung involvement are of value. The lung will in some cases show a general bronchitis (tuberculous miliary process) or an area of apex consolidation. In other cases, especially in young infants, the lung may present no physical signs.

*General Symptoms.*—Of great value is the slow insidious onset, followed abruptly by vomiting and convulsions with subsequent unconsciousness.

*Vomiting* sets in on the average eighteen days before the fatal issue and after the onset of premonitory symptoms, and may occur once or twice daily. It is absent in some cases. Localized convulsions may appear two weeks after the initial attack of vomiting.

These symptoms in infants and young children in association with a fever of slight or moderate intensity, and bulging of the fontanelle (if it is still open), are valuable aids to diagnosis.

The slow, irregular pulse and the respiratory phenomena are present in other forms of meningitis. The cerebral cry (cries hydrocephalique) is of confirmatory value only. The emaciation and retracted abdomen, as well as the opisthotonos and the bulging fontanelle, may be present in other forms of meningitis, such as the cerebrospinal meningitis of the epidemic type, and especially in that form described by English authorities as posterior basic meningitis. The symptoms of optic neuritis are present in cerebrospinal meningitis as well as in the tuberculous form. In the latter, they are sometimes absent.

The choroid is sometimes the seat of tubercle deposit. Choroid tubercle was seen during life in 3 out of 30 of my cases.

Of great service in making a clinical diagnosis is the presence of palsies of the cranial nerves. Ptosis, facial paralysis, strabismus, paralysis of the internal rectus of one side, or ptosis on one side and lagophthalmus on the opposite side, are indicative of a lesion at the base of the brain. These palsies are seen much more frequently in the tuberculous forms of meningitis than in cerebrospinal meningitis of the epidemic type. I have, however, seen them in cases of cerebrospinal meningitis in infants under the age of twelve months. If the lateral ventricle is distended with fluid, the hollow note

described by Macewen may be elicited by percussion along the parietal or frontal bone. As the infant lies in bed with the head supported on its side the note is best obtained by gentle percussion on the frontal or parietal bone lying inferiorly. It may also be obtained on the superior frontal or parietal bone. I have found this sign of great utility in the early stages of the disease.

Lumbar puncture is to-day the most valuable aid in making a diagnosis of tuberculous meningitis. We must not expect to find tubercle bacilli in all fluid withdrawn, for in certain cases they are so few in number as to escape detection. In the majority of my cases they were present. Fürbinger found bacilli in 27 out of 37 cases. I have come to look upon certain negative characteristics of the puncture fluid as of great value. The fluid is clear or shows slight turbidity in strong light, owing to the presence of a fine, dust-like formation. The absence of all bacteria or leucocytes in these clear or turbid fluids and the formation in the test-tube after twenty-four hours of a peculiar funnel-shaped, cobweb-like structure, point strongly to the tuberculous nature of the fluid.

Tuberculous meningitis must be differentiated from epidemic and sporadic cerebrospinal meningitis (Weichselbaum), apex pneumonia, typhoid fever, sepsis, disturbances of the stomach and gut, uræmia, helminthiasis, and disease of the ear.

The onset of epidemic cerebrospinal meningitis is acute and the cerebral symptoms develop rapidly. The fluid obtained by lumbar puncture will show the intracellular diplococci even if clear. In the majority of cases the fluid is turbid after the fifth day of the disease.

Pneumonia with cerebral symptoms may simulate tuberculous meningitis. Here again the history and the character of the delirium in older patients will aid us. The signs in the lung and the presence of leucocytosis, which is marked in pneumonia and generally absent in tuberculous disease, are significant. I have, however, seen cases of tuberculous meningitis with leucocytosis. In the majority of cases of typhoid fever the history will be of service in connection with the roseola, the Widal reaction, the enlarged spleen, and the absence of leucocytosis. Diarrhœa may be present in typhoid.

Disturbances of the gut, uræmia, and helminthiasis may present symptoms resembling those of tuberculous meningitis, but the symptoms in time retrograde or are cleared up by a study of the case.

I have seen otitis media in nurslings with very limited areas of bronchopneumonia, simulate tuberculous meningitis. In these cases the infants may have been ill for two weeks or more. They start from sleep, are irritable on awakening, and lose appetite.

In one case the ocular symptoms closely simulated those of tuberculous meningitis. As a rule there are intervals during which the child is not only free from pain, but also has a normal temperature. At other times the temperature has a septic intermittent character,



and mounts higher (104° F., 40° C.) than in tuberculous meningitis. Aural examination only will remove doubt.

The **duration** of the disease varies within wide limits; I have seen cases which extended over three months. The majority of cases last from two to three weeks, but cases lasting five weeks are not unusual. The very rapid cases in which death ensued within twenty-four hours have been mentioned.

The **prognosis** is always fatal. Isolated cases of recovery have been reported, but should be regarded with doubt.

The **treatment** is directed to alleviating the sufferings of the patient. Lumbar puncture is not curative, and should not be repeated after the first diagnostic puncture has been performed.

### **Tuberculosis of the Brain.**

*(Solitary Tubercle of the Brain.)*

In this there may be a single localized tuberculous nodule or mass in the brain, or several such formations may be present. Demme found a growth of this kind in an infant twenty-three days old. Hensch has published a case in an infant eleven days old. The majority of cases occur between the second and the fifth year.

**Morbid Anatomy.**—Tubercle bacilli of diminished virulence and limited number are carried from the focus of tuberculosis to the brain through the blood-channels, and there lodged in a terminal bloodvessel, forming solitary tuberculous masses varying from the size of a pea to that of a hazelnut. These are surrounded by a zone of granulation-tissue. The neuroglia in the immediate vicinity is the seat of proliferation, and may form a capsule around the growth. Circumscribed meningitis over the situation of the growth, with adhesions of the pia mater to the dura, may be present. Fully half of these solitary growths occur in the cerebellum (Gerhardt). The growth may be single or there may be one large growth and several of smaller size. Starr and Seidl found a solitary growth in 77 per cent. of the cases. The larger number of brain tumors in infancy and children are tuberculous. Starr found this variety in 152 out of 300 cases of all kinds of tumors.

The symptoms are those common to all tumors, and will be described in the section devoted to Brain Tumors.

## **SYPHILIS.**

### **Acquired Syphilis of Infancy and Childhood.**

**Etiology.**—Of 42 cases of acquired syphilis collected by Fournier, 19 were infected by the father or mother after birth, and 8 by the nurse. No case was infected in passing through the maternal parts, and no infant was infected by the mother if she had

contracted the disease just prior to her accouchement. A child of a syphilitic mother, if born free from signs of syphilis, cannot contract a primary lesion at birth from the maternal parts, even if these parts are the seat of condylomata, nor can such an infant be infected subsequent to birth. It has an acquired immunity against the disease.

A chancre or primary lesion is, in the infant as in the adult, the only evidence of acquired syphilis. It is the result of infection, and must be present in order that the diagnosis may be certain. Chancres are rarely genital. They are found, as a rule, in the mouth, on the face, and on the abdomen and perineum. An infant may be infected by the nipple of the nurse's breast. The act of kissing, contaminated nipples of the nursing-bottle, instruments, sponges, ritual circumcision, and humanized vaccine virus, are all means of infecting the infant. Since humanized vaccine virus is no longer used, this mode of infection has been eliminated.

The **symptoms** consist of a chancre or initial lesion, rarely genital, which appears three or four weeks after inoculation. The other accidents, such as bubo or adenopathies, the eruption, and all the secondary symptoms of acquired syphilis, appear in due course as in the adult. The genital chancre is seen in infections caused by ritual circumcision.

The **prognosis** as to life is good in comparison with that in the hereditary form of the disease. While in the hereditary form the mortality is from 70 to 80 per cent., that in the acquired form is very low. Fournier lost only 1 in 42 cases of acquired syphilis. The course in infants and children is benign. The chancre is not well developed; the induration is present only a short time, or may even escape notice. The infants enjoy good health in spite of the presence of the secondary symptoms. I have confirmed these statements by observing 7 cases of genital chancre. The tertiary manifestations, such as gummata, bone lesions, joint-affections, eye and laryngeal symptoms, and cerebrospinal lesions, appear from five to twenty-five years after the initial lesion.

**Differential Diagnosis.**—Acquired syphilis must be differentiated from the hereditary form of the disease. Hereditary or congenital syphilis appears early without an initial lesion, showing general secondary symptoms from four to six weeks after birth. The chancre is the first manifestation in acquired syphilis. In Fournier's 42 cases the chancre appeared during the first year of life in 19, and during the second year in 10 cases. The snuffles, pemphigus, and pseudoparalysis are not present in acquired syphilis. Secondary accidents, such as mucous patches or papules about the genitals, appearing during later childhood are probably traceable to a post-natal infection. Interstitial keratitis, bone syphilis, and cutaneous stigmata are common to the hereditary and acquired forms of the



disease. It is sometimes very difficult to decide which form of the disease is present. Thus far no one has shown conclusively that Hutchinson's teeth are present in acquired forms of syphilis in infancy and childhood. Their presence is therefore strong presumptive evidence of hereditary syphilis.

### Late Hereditary Syphilis.

(*Syphilis Hereditaria Tarda.*)

Fournier defines late hereditary syphilis as a symptom-complex of accidents of syphilis originating in a hereditary infection, which manifest themselves at a more or less advanced period of life, that is to say, in the majority of cases between the third and the twenty-eighth year. There are two classes of cases. In the first, the patient has remained in perfect health without any of the eruptive or other symptoms of hereditary syphilis until at an advanced period of childhood one or more of the symptoms of late hereditary syphilis are developed. In the second, the late symptoms have been preceded by the early symptoms of hereditary syphilis. The late symptoms may develop after an interval of from ten to fifteen years. The cases of the former class have been the subject of much discussion. The occurrence of the second class of cases is now well established; it is often very difficult to determine the hereditary or acquired nature of the original infection.

Fournier, in classifying the symptoms of 212 cases of late hereditary syphilis, found the eye to be the organ most frequently affected. Next in order of frequency are the lesions of the bones and skin. The rarer affections are those of the kidney, larynx, spinal cord, testes, and lungs.

The subjects of late hereditary syphilis have certain well-defined general characteristics. They are constitutionally delicate and have an emaciated habitus. The skin presents a grayish anæmia. There is an arrest in the development of bone and musculature. The men are undersized and present the picture which has been characterized as infantilism. The signs of virility, such as the beard, hair under the arm and on the pubes, are scantily developed. The testes are rudimentary. The adult has the appearance of a boy of fourteen or fifteen years. The women are correspondingly backward in development.

**The Eye.**—The eye symptoms appear most frequently at the age of ten or fifteen years, but may become evident as early as the third year. The principal symptom is a keratitis of the diffuse interstitial variety, the so-called keratitis of Hutchinson. The cornea has a slightly cloudy or filmy appearance, or the whole structure is diffusely opaque. The other ocular accidents are plastic iritis, which fixes the iris, thus limiting its action and causing a difference in the

size of the pupils. The rarest manifestations are miliary gummata of the iris.

The **bone-lesions** are most frequent between the fifth and the twelfth year.

The *head* presents a cuboidal shape; the forehead is prominent; the frontal bones have large bosses, as have also the parietal bones. The longitudinal suture is depressed, giving a natiform shape to the head. The cranium may have the form seen in mild degrees of hydrocephalus.

The *nose*, on account of the destruction of the bony septum, has a depressed bridge. The bony and cartilaginous septa form an acute

FIG. 74.



Late hereditary syphilis; bone deformity and sinus. Child, three years of age.

angle, and a peculiar *retroussé* appearance is given to the organ. Both bony and cartilaginous septa may be destroyed. The whole organ is flattened, the tip of the nose being wrinkled into three or more folds.

The *long bones* are especially affected by the accidents of late hereditary syphilis, the tibia being most frequently affected. The lesion may consist in an osteoperiostitis, a gummatous osteoperiostitis, or a gummatous osteomyelitis.

If osteoperiostitis is present, there are diffuse swelling and thickening of the bone—the so-called *sabre-like deformity* (Fig. 74). This process may affect the long bones of the upper extremities. The gum-



matous lesions of osteoperiostitis form numerous irregular painful swellings on the bone. Gummata are present on the flat bones of the cranium. When these break down, the destructive processes may expose the dura mater. Arthropathies with synovitis may be mistaken for tuberculosis of the joint. This form of synovitis is generally bilateral. One of my cases, a child five years of age, gave no history of syphilis. The radius on both sides was affected by osteoperiostitis. The joints may be deformed by osteophytic growths involving the epiphysis or head of the bone.

The ear is affected by an otitis with destruction of the ossicles, and even by mastoid disease. In other cases deafness supervenes without premonitory symptoms.

The skin and mucous membranes show certain stigmata in the form of cicatrices of recent or old ulcerations. These may exist on any part of the body, but are especially characteristic on the vermilion border of the lips and at the corners of the mouth, where they are seen as radiating, linear pale-white fissures.

The lymph-nodes may be enlarged, especially those on each side of the neck, below the jaw, and in the axilla and inguinal regions.

The spleen is enlarged, but not so frequently as is stated by some authors. Fournier found it enlarged in 15 out of 212 cases.

The liver was enlarged in 25 cases. In one of my cases of late hereditary syphilis in a child eight years of age, post-mortem examination revealed cirrhosis of the liver of the hypertrophic type. There were enlargement of the spleen, icterus, and ascites; Hutchinson's teeth were well marked, and there were also adenopathies and vasculitis.

Fournier among others has described forms of idiocy and epilepsy of syphilitic origin, but there is great difference of opinion on this question. The theory of Parrot, that rachitis is the result of syphilis, is now generally abandoned. The deformities of the teeth which occur in late hereditary syphilis will be found fully described in the section devoted to Dentition.

### **Congenital or Hereditary Syphilis.**

Congenital or hereditary syphilis results from the infection of the ovule or fœtus in utero. This may occur in a number of ways, but in the great majority of instances it results from infection of the fœtus through the father. The more recent the syphilis of the father, the more likely is the infection to occur. It is most certain to occur if both the father and mother suffer from recent syphilis at the time of conception. The father may at the time of insemination suffer from recent syphilis and the mother be healthy. Under such conditions the child is born syphilitic. The mother may not show any signs of active syphilis either during pregnancy or at any subse-

quent period. The mother may suckle her offspring, which shows all the marks of active hereditary syphilis, without becoming infected, but the child will infect any strange nurse. The mother has during pregnancy acquired an immunity against the infection. This phenomenon, which is a matter of daily observation, was first brought to the notice of the profession by the distinguished surgeon Colles, and has since become known as Colles's law. The longer the mother is subjected to the influence of the syphilitic virus, the more permanent does her immunity become. Thus a mother who has at first miscarried may eventually give birth to a living infant which bears the marks of syphilis. As the virus becomes weakened, the mother may bear an infant to all appearances healthy. In the interval, although repeatedly pregnant, the mother has shown no signs of active syphilis.

If the father is healthy at the time of insemination and the mother the subject of recent syphilis, the infant will be born syphilitic. On the other hand, if the mother contracts syphilis after conception, the father at the time of conception having been healthy, the infant may or may not be born syphilitic. The nearer the time of the infection of the mother to the end of her period of pregnancy, the more likely is the infant to escape (Monti, Zeissel, Hutchinson). Such an infant if born healthy may become infected in the ordinary way from the mother after birth.

A father who has passed through the secondary manifestations of syphilis may in the late secondary period or tertiary stage fail to convey the poison in the sperma. The result will be an infant free from syphilis (Fournier, Neuman). Yet so far-reaching is the influence of the syphilitic dyscrasia that such an infant, although born healthy and at no time showing signs of syphilis, may present certain signs, such as peculiarities of bone formation (teeth) traceable to the syphilitic virus (parasyphilitic).

Exceptions to Colles's law occur, as is to be expected. Fournier has recorded cases in which mothers apparently immune have developed signs of secondary syphilis after the birth of the infant. Finger has met cases in which tertiary syphilis developed in the mother subsequent to pregnancy without the occurrence in her of any of the signs of secondary syphilis.

Of 218 mothers who had borne syphilitic infants, Hochsinger found 72 who were free from manifestations of secondary or tertiary syphilis although observed for years.

**Morbid Anatomy.**—In considering the pathology of hereditary syphilis, Hochsinger divides the cases into four classes:

The first class of cases die in utero before the eighth month, Autopsies upon such fetuses show general parenchymatous involvement of the glandular apparatus with epiphyseal osteochondritis.

The second class includes infants born living or dead before the



end of pregnancy. They present at birth a papulobullous syphilide. In these cases diffuse parenchymatous changes are found in the viscera, and frequently marked epiphysitis.

The third class comprises infants born living and without any exanthema, but which later develop an exanthema independently of visceral or bony changes.

The fourth class comprises infants born without an exanthema, but having at birth marked visceral and bone-changes.

Taking up in detail the lesions found in the various parts of the body, we find that the *skin* shows an increase in the thickness of the rete Malpighii, caused by swelling of the cells of the rete, serous infiltration of this layer, and an increase of the spaces between the cells of the rete. The horny layer of the skin is much thinned in comparison, although there is a constant throwing-off of the cells of this layer in lamellæ. The epithelium of the sweat-glands is swollen and there is a small round-cell infiltration between the glands. There is a vasculitis of the small bloodvessels affecting the external coat chiefly. Pemphigus and bullæ result from infiltration of the rete and the lifting up and separation of the horny from the papillary layer by serum.

**The Lungs.**—The changes in the lungs may be considered under two heads :

First, the lungs of infants born dead or who have died soon after birth, are collapsed, devoid of air, hyperæmic, and dark red in color. In rare cases the lungs may be diffusely whitish yellow in color, giving the appearance of the so-called pneumonia alba. The second class comprises infants that have breathed, and that show a gray or grayish-white discoloration of the lungs in places. There is residual air in the lungs, and they are denser and larger than is normal.

Ziegler has shown that the changes in the lungs consist chiefly in an increase in the interalveolar connective tissue, the formation of new vessels, and vasculitis of the bloodvessels. In the majority of newly born infants the alveolar epithelium is but little affected. In pneumonia alba there is a proliferation of the alveolar epithelium, giving a peculiar appearance and color, hence the name.

**The Liver.**—Changes in the liver are quite constant in hereditary syphilis. These may or may not be associated with enlargement of the organ. Out of 148 cases of congenital syphilis, Hochsinger found the liver enlarged in 46 ; in all but 2 the spleen also was enlarged ; in the severer cases the liver was markedly so.

The pathological changes in the liver have been described by Hudelo, Hochsinger, and Heller. There may be simply diffuse, small round-cell infiltration of the interstitial connective tissue, with inflammatory changes in the smaller arteries. The liver in these cases is not enlarged. In the cases presenting an enlarged liver there is interacinous proliferation of connective tissue, beginning at

the periportal region and following the course of the bloodvessels. There is *vasculitis*, shown in a thickening of the adventitia of the bloodvessels. The parenchyma is degenerated. In other cases interminous collections of small round cells are on gross sections of the liver seen as yellow pinhead-sized spots. These are called by Hochsinger *miliary gummata*. Fully developed gummata of large size are very rare in the liver of infants affected with hereditary syphilis.

The spleen is in some cases enlarged to ten times its normal size. Gummata, single or multiple, occur, but are rare. In hereditary syphilis not only is the parenchyma increased, but also the connective tissue of the spleen.

**Kidneys.**—In rare cases there are induration and contraction of the kidney. The parenchyma is retarded in development by intra-uterine syphilis and the connective tissue increased.

The pancreas may be enlarged and infiltrated, the parenchyma hard, and the interstitial connective tissue increased. There may be condylomatous ulcerations on the tongue, pharynx, and tonsil.

According to Hochsinger, the *glandular apparatus* of the gut may show a diffuse small-cell infiltration, Peyer's patches may be infiltrated, and the vessels may be the seat of a vasculitis. The lymph-nodes are, as a rule, little changed except in cases with late manifestations. The thymus gland in cases of hereditary syphilis has been found to be the seat of cystic degeneration (Eberle, Ribbert), caused by the dilated epithelial spaces of the fetal thymus.

**Bone-changes.**—The bone-changes in hereditary syphilis occur principally at that part of the bone between the epiphysis and diaphysis in the lower end of the femur, tibia, and radius. In the milder forms of bone-change there is, according to Ziegler, little real inflammation. There are irregularity in the deposit of lime salts and the formation of marrow-spaces. In severe forms there is a true inflammatory process. In the vicinity of the joint-cartilage, grayish-red, yellowish-white, or yellowish-green foci of osteomyelitis are found. The irregular deposit of lime salts and the formation of marrow-spaces are evidenced by reddish-yellow projections of marrow-spaces into the adjacent proliferated cartilage. These give the epiphyseal junction a more irregular and widened appearance than is normal. Sometimes separation of the epiphysis at the junction of the diaphysis occurs. The above changes are frequent, although not constant. In the later stages of syphilis in children there are, as in the adult, caries, necrosis, and gumma formations in the long and the cranial bones.

**Symptoms.**—The symptomatology of hereditary syphilis varies largely with the class of cases. In some cases the fetus is expelled dead, bearing the marks of fully developed syphilis in the shape of skin, bone, and visceral lesions. In others the infant is born living,





the periportal region and following the course of the bloodvessels. There is vasculitis, shown in a thickening of the adventitia of the bloodvessels. The parenchyma is degenerated. In other cases inter-acinous collections of small round cells are on gross sections of the liver seen as yellow pinhead-sized spots. These are called by Hochsinger miliary gummata. Fully developed gummata of large size are very rare in the liver of infants affected with hereditary syphilis.

The **spleen** is in some cases enlarged to ten times its normal size. Gummata, single or multiple, occur, but are rare. In hereditary syphilis not only is the parenchyma increased, but also the connective tissue of the spleen.

**Kidneys.**—In rare cases there are induration and contraction of the kidney. The parenchyma is retarded in development by intra-uterine syphilis and the connective tissue increased.

The **pancreas** may be enlarged and infiltrated, the parenchyma hard, and the interstitial connective tissue increased. There may be condylomatous ulcerations on the tongue, pharynx, and tonsil.

According to Hochsinger, the **glandular apparatus** of the gut may show a diffuse small-cell infiltration, Peyer's patches may be infiltrated, and the vessels may be the seat of a vasculitis. The lymph-nodes are, as a rule, little changed except in cases with late manifestations. The thymus gland in cases of hereditary syphilis has been found to be the seat of cystic degeneration (Eberle, Ribbert), caused by the dilated epithelial spaces of the foetal thymus.

**Bone-changes.**—The bone-changes in hereditary syphilis occur principally at that part of the bone between the epiphysis and diaphysis in the lower end of the femur, tibia, and radius. In the milder forms of bone-change there is, according to Ziegler, little real inflammation. There are irregularity in the deposit of lime salts and the formation of marrow-spaces. In severe forms there is a true inflammatory process. In the vicinity of the joint-cartilage, grayish-red, yellowish-white, or yellowish-green foci of osteomyelitis are found. The irregular deposit of lime salts and the formation of marrow-spaces are evidenced by reddish-yellow projections of marrow-spaces into the adjacent proliferated cartilage. These give the epiphyseal junction a more irregular and widened appearance than is normal. Sometimes separation of the epiphysis at the junction of the diaphysis occurs. The above changes are frequent, although not constant. In the later stages of syphilis in children there are, as in the adult, caries, necrosis, and gumma formations in the long and flat cranial bones.

**Symptoms.**—The symptomatology of hereditary syphilis varies largely with the class of cases. In some cases the foetus is expelled dead, bearing the marks of fully developed syphilis in the shape of skin, bone, and visceral lesions. In others the infant is born living,





PLATE XIV.



Congenital Syphilis. Showing nasal deformity.  
Newborn infant.



but presents a few very characteristic signs of syphilis, such as the presence of bullæ or pemphigus either on the palms or on the soles of the feet. The vesicles may be filled with a purulent fluid. As a rule these infants are emaciated. In some cases the bridge of the nose is sharply depressed and forms a distinct angle with the cartilaginous septum (Plate XIV.). This intra-uterine deformity in the newborn infant has been studied by Epstein. Such infants suffer from a troublesome coryza and cannot breathe freely through the nose. They present enlargement of the liver and spleen, and there may be a few copper-colored discolorations on the skin of the forehead and nose. The lips have a shiny, glossy appearance, and after a time may present distinct rhagades. Some days after birth there is a diffuse syphilitic eruption of papules or vesicopapules, with the so-called diffuse induration of the skin of the palms of the hand and soles of the feet, described by Hochsinger. Here and there discolored spots which were formerly mistaken for papules may be seen. The skin of the face may have a diffuse coppery color. Patches of discolored skin appear and become confluent, the coryza and rhagades along the lips and at the angle of the mouth become more marked, and the rhagades bleed easily.

In another class of cases the infant is born well nourished and has a good color. Within from two to four weeks a general eruption of papules and vesicopapules appears. Some of the vesicopapules are purulent, and after bursting dry up, leaving the surface covered with crusts on a copper-colored base. In these cases the manifestations on the mucous membranes, including coryza and rhagades, are also gradually developed (Fig. 75).

If the above symptoms are marked, we may find enlargement of the liver and spleen. I have seen the most marked signs of hereditary syphilis of the skin without the slightest enlargement of the liver or spleen. As a rule the arms will present papules, which may ulcerate at the points of contact with adjacent surfaces of skin. The typical condyloma lata is not frequent in early hereditary syphilis. The nates have a coppery shining color, are cracked in places and diffusely indurated (Hochsinger's induration). The trunk may present few symptoms. The bicipital glands are enlarged if the syphilitic exanthema is fully

FIG. 75.



Hereditary syphilis: rhagades and mucous patches of upper and lower lips.

developed. The thighs show brownish, copper-colored patches. These patches give the skin a marbled appearance, which differs from that of the so-called healthy marbled skin in that the discolored areas are surrounded by normally colored skin, while in ordinary marbled skin the opposite condition obtains. On exposed areas, such as the knees, nates, soles of the feet, and palms of the hands, the skin is diffusely indurated.

In a detailed consideration of the lesions, those of the skin are the first to engage attention. The most common forms of eruption are the papular or the papulopustular form of syphilide. This may be combined with the macular form; in fact, it is common to find in the same case all forms in various stages of development.

FIG. 76.



Congenital syphilis: circinate syphilide of the nose.

The papules occur on the forehead, palmar surface of the hands and plantar surface of the feet, and on the nates (Fig. 76). They show a distinct induration of the skin, are raised above the surface, and have a glossy, copper-colored appearance. On the nates or in the groin the papules may ulcerate; very rarely these form condylomata lata in the early periods of congenital syphilis. The condyloma is a feature of the later period of this disease. Macules develop within the first three months of life, and from the sixth to the tenth week are associated with seborrhœa. Infants thus affected are born with a peculiar anæmia, in which the skin has a cadaveric hue. The macules appear on the forehead and face as copper-hued spots, which increase in number until the skin has a general marbled appearance



PLATE XV.



Mucous Patches or Flat Condylomata in a child twenty months of age.  
Congenital syphilis.





(*roseola syphilitica*). They then fade, leaving the surface covered with brownish-red areas. These persist around the *alae nasi* and the forehead for a long time, giving the face a peculiar dirty-yellow spotted appearance.

The diffuse syphilitic infiltration of the skin has been studied by Hochsinger. It is not the forerunner or the sequence of any papular eruption. It appears in the third week in 50 per cent. of the cases, and reaches its height between the eighth and the tenth week. It first presents discolored areas on the palms and on the soles of the feet, on the nates, the calves of the legs, also on the cheeks and chin, where it forms rose-colored or copper-colored areas which coalesce. The soles and palms may appear diffusely red or bluish and glossy. The skin is diffusely thickened on the palms and soles and desquamates in lamellæ. At the junction of the mucous membranes and skin fissures result on account of the thickening of the skin. The lips appear anæmic as a result of the infiltration of the mucous membrane, and are fissured. There are rhagades at the *alae nasi*. The rhagades at the angles of the mouth are covered with a bluish-white pellicle, and the surrounding skin is copper-colored. There are swelling of the nasal mucous membrane and a thin, purulent discharge mixed with blood. The hair falls out on account of the infiltration of the scalp; the scrotum is thickened and fissured from the same cause.

The blood shows all stages of anæmia, from the mildest to the grave pseudoleukæmic anæmia of von Jaksch, which some authors trace to syphilitic influences.

The bones are affected with an osteochondritis, already described. This may appear in the first few weeks or at a much later period. It manifests itself by pain in moving the joints. The infant cries when handled. The mother notices that one arm lies motionless at the side, and that every attempt to move it causes pain. Parrot described this condition as a pseudoparalysis. At the junction of the epiphysis and diaphysis at the lower end of the humerus or radius the bone may be swollen and painful. As a rule, the process affects the upper extremity on one side only, but in severe cases both the upper and lower extremities may be involved. In some cases this symptom may be present without a skin eruption. The other conditions which simulate it are septic osteomyelitis involving the joints, and severe congenital rachitis. I have known instances in which prolonged observation was necessary to clear up the case.

A very characteristic but not common affection of the bones is the so-called dactylitis syphilitica (Fig. 77). This may appear as early as the fourth week, and may be associated with swelling of the epiphyses of the long bones. It consists of a fusiform swelling of the phalanges of one or more fingers. According to Taylor, this is primarily a gummatous infiltration of the skin, the periosteum, bone,

and epiphyseal cartilage becoming involved. In another form the periosteum and the bone itself are the seat of the gummatous inflammation, the epiphysis and the joint becoming involved later in the process. In neglected cases, fistulae and destruction of the joint may result from necrosis of the epiphysis. The diagnosis of these forms of dactylitis from tuberculous spina ventosa is sometimes difficult, and often impossible without mercurial treatment. I have lately seen a case of rachitis which involved the phalanges of all the fingers, and which simulated very closely the above affection (see Rachitis).

Syphilitic affection of the liver gives no symptoms. Hensch records cases in which icterus was associated with enlargement of

FIG. 77.



Congenital syphilis: onychia of all the nails; dactylitis of the phalanx of the index finger. Infant, four months of age.

the organ. Hochsinger denies the occurrence during the nursing period of any authentic case of syphilis of the liver with icterus or ascites.

Somma, Fischl, and Kohts have described symptoms of cerebral syphilis in infants that were subjects of hereditary syphilis. Convulsions, hydrocephalus, epilepsy, and paralysees have been traced to the presence of gummous meningitis or sclerosis. That such changes occur as a direct result of syphilis at so early a period is doubted by Hensch. I have not seen manifestations of cerebral syphilis in infants. Hensch is also inclined to include Mrazek's cases of



hemorrhagic syphilis among the septic diseases of the newborn occurring in syphilitic infants.

Antonelli in 1897 described changes in the fundus oculi of newborn syphilitic infants. These consisted of optic neuritis, retinitis vascularis, and retinochoroiditis. He believes these changes to be causative in the production of myopia and strabismus in such infants.

The **diagnosis** of hereditary syphilis is not difficult in the vast majority of cases. If the fœtus is expelled dead, it bears the marks of syphilitic infection, such as bullæ and affections of the internal organs. Maceration alone is not indicative of syphilis. If

FIG. 78.



Hereditary syphilis: gummata of the cranial bones. Child, eighteen months of age.

the infant is born living, the evidences of syphilis are sometimes very few and equivocal. After a few months the diagnosis will sometimes be difficult; the eruption will have disappeared, leaving only an anæmia of uncertain origin, with a few discolored areas about the nasolabial folds and around the temporal region. There is a suspicious dirty-looking seborrhœa of the supra-orbital region. A rebellious anal eczema or copper-colored intertrigo which resists treatment should arouse suspicion. Pustular papules are not pathognomonic even if combined with joint-affections. A case came under my notice in which an infant had a varicella-like eruption with a painful swelling of the right elbow-joint. A diagnosis of epiphysitis syphilitica had been made and the eruption had been mistaken for a syphilide. The color of the eruption was not that of a syphilide. Expectant treatment and immobility of the joint proved, after a few days, that the case was one of varicella with the joint-complication sometimes seen in that disease.

In the diagnosis of late hereditary syphilis the symptomatology

is of service. In cases with bone-lesions it is often very difficult to differentiate it from tuberculous affections (Fig. 79). An active course of treatment then becomes necessary, with a view to diagnosis. This is especially the case in arthropathies, and also in late forms of dactylitis.

The **prognosis** as to life depends upon several factors. A breast-fed infant is more likely to survive than a bottle-fed infant. The possibility of complete restoration to the normal is slight. The majority of infants bear the marks of the disease into adult life, even under very favorable conditions of treatment and environment, and develop late in life the so-called late symptoms of hereditary syphilis. Some infants while progressing favorably under treatment, die suddenly without apparent cause; others remain stunted and

FIG. 79.



Tuberculous affection of the bones of the hand simulating syphilitic disease. Child, sixteen months of age.

delicate throughout childhood. Rachitis and its sequelæ seem to be very prevalent among infants that are the subjects of hereditary syphilis.

The **treatment** of congenital syphilis may be either internal or by inunctions or injections. I have found internal treatment to be the more satisfactory. The effects of the mercury are not so injurious as is the case with the inunction methods. The drug employed was calomel in combination with the saccharated ferric carbonate (this was a favorite remedy of Widerhofer):

Calomel . . . . .	gr. $\frac{1}{2}$ (0.01).
Ferri carb. sacc. . . . .	gr. iij (0.18).
Ft. pulver.	

A powder of this size may be given every three hours or four times a day. Some authors (Baginsky) prefer the protoiodide of mercury, grain  $\frac{1}{8}$  to  $\frac{1}{2}$  (0.01 to 0.03). If there is intolerance to calomel,



satisfactory results may be obtained by the use of Lustgarten's preparation of hydrarg. oxydulatum tannicum, in doses of grains *ij* to *v* (0.1 to 0.3), repeated every three hours or four times daily.

If the rhagades, especially those about the anus, bleed or heal slowly, they should be stimulated with a weak solution of silver nitrate. Calomel should be dusted upon condylomata lata three times daily.

Baths of sublimate are recommended in severe cases of pemphigus, but it is not often necessary to resort to them.

Infants in the nursing period do not bear inunctions well. I have seen several cases treated by this method which lost weight rapidly or died suddenly, and this has been the experience of others (Monti). The old method was to place grains *vij* to *xv* (0.5 to 1.0) of unguentum hydrarg. under the flannel abdominal binder daily, and allow it to be absorbed, or the same quantity of ointment was rubbed in daily on various parts of the body.

Severe rhinitis is best treated by washing out the nasal passages once a day with a solution of corrosive sublimate (1 : 2000). The small glass syringe with a blunt soft-rubber nozzle is best for this purpose. After the syringing, unguentum iodoform. is applied to the interior of the nose by means of a camel's hair pencil.

How long should treatment be continued? No matter what method of treatment is adopted, mercury should be administered until all discoloration of the skin has disappeared. To attain this result will take a varying length of time in different cases. After the skin is clear and the anæmia has disappeared, it is well to cease the administration of drugs and observe the patient for further symptoms. Sometimes a patient will be brought to the physician for the treatment of a rebellious intertrigo long after all signs of general syphilis have disappeared. Such an intertrigo may have a copper color, and may ulcerate, the ulcers having a peculiar lardaceous appearance. In these cases, even if all other signs of congenital syphilis are absent, the internal administration of mercury gives brilliant results.

The treatment of late hereditary syphilis will depend much upon the nature of the therapeutic measures adopted earlier in life. In the majority of cases, the subjects being in later childhood or adolescence, it is well to begin treatment by a full inunction course, conducted on the same plan as with adult subjects with acquired syphilis. In addition, if gummatous affections of the bones are present, and if as in one of my cases visceral lesions, such as enlargement of the liver, have appeared, the patient is put upon gradually increasing doses of iodide of potassium. In one of my cases large doses of iodide of potassium failed to relieve the intense headache. This patient married, and after having a miscarriage gave birth under specific treatment to a healthy infant. The treatment of

acquired syphilis does not differ from that of congenital or late hereditary syphilis.

#### LEADING AUTHORITIES REFERRED TO IN CHAPTER III.

- Anton W.*: Die diphtherie der nase, Jena, 1897.  
*Boginsky, A.*: "Diphtherie," etc., Nothnagel's Spec. Path. u. Therap., 1898, Bd. II., I. Theil.  
*Behring, E.*: Therapy of Infectious Diseases, Parts I. and II., Berlin, 1900 (allgm. therapie der infec. Krankheit).  
*Blackader, A. D.*: "Relation between Human and Bovine Tuberculosis," Boston Medical Journal, Dec., 1901.  
*Blumer, G.*: "Infection in Infants due to the Typhoid Bacilli," Trans. Amer. Med. Assoc., 1900.  
*Celli, A.*: Malaria, Vienna, 1900. Beitr. zur exper. Therapie.  
*Cornet, G.*: "Die Scrofulose," Nothnagel's Spec. Pathol., Bd. xiv.  
*Councilman, Mallory and Pearce*: Bacteriology and Pathology of Diphtheria, Sears Lab. Rep., 1901.  
*Councilman, Mallory and Wright*: Report State Board of Health of Massachusetts, 1898.  
*Dennig, A.*: Tuberculose im Kindesalter, 1896.  
*Duel, A. R.*: "Acute Otitis Media and Mastoiditis in Scarlet Fever and Measles and Diphtheria," Review of Reviews, 1901.  
*Fielder, F. S.*: "Vaccination," Medical News, 1901.  
*Fournier, A.*: Syphilis hereditaire tardive, Paris, 1886.  
*Gaessler*: Mittellorrhoe bei Scarlatina, 1900.  
*Gershel, M.*: "Value of Widal Reaction," etc., Med. Rec., Nov., 1901.  
*Herzfeld*: "Tubercul. Peritonitis," mittheilungen aus dem grenzgebiet der med. 1901.  
*Hochsinger, C.*: Hereditare Syphilis, Vienna, 1898.  
*Koplik*: "Cerebrospinal Meningitis," Medical News, 1901.  
*Koplik*: "Malaria in Infants and Children," New York Medical Journal, 1899.  
*Koplik*: "Measles Spots," Archives of Pediatrics, 1896.  
*Macewen*: Pyogenic Diseases of the Cord, 1893.  
*Mattié, L.*: Sérotherapie preventive de la diph., 1901.  
*Morse, J. L.*: "Widal Reaction," Archives of Pediatrics, 1901.  
*Netter*: "Meningitis," Transactions International Medical Congress, 1900.  
*Nobling-Jankau*: R. Fischl in Prophylaxis der Krankheiten des Kindesalters, 1900.  
*Pearce, R. M.*: "Scarlet Fever," Medical and Surgical Reports, Boston City Hospital, 1899.  
*Pearce, R. M.*: "Bac access sinuses scarl. and diph.," Journal Boston Society Medical Sciences, 1899.  
*Renaud*: Thèse, Leucocytosis in Measles, Paris, 1900.  
*Schmidt*: "Rötheln," etc., Wien. klin. Wochenschr., 1900.  
*Shaw, H. L. K.*: "Prophylaxis Antitoxin," Albany Medical Annals, 1901.  
*Slawyk*: "Koplik's Spots," Deutsche med. Wochenschr., 1898.  
*Thursfield, H.*: "Posterior Basic Meningitis," Lancet, 1901.  
*Wentworth*: "Meningitis in Infants and Children," Boston Medical and Surgical Journal, 1898.  
*West, J. P.*: "Glandular Fever, with Literature," Archives of Pediatrics, 1899.



## CHAPTER IV.

### DISEASES OF THE MOUTH, PHARYNX, AND LARYNX.

#### THE CHARACTERISTICS OF THE NORMAL MOUTH.

THERE are certain localities in the mouth which are particularly liable to aphthæ and ulceration. The mucous membrane over the hamular processes of the palate bone presents whitish areas paler than the surrounding mucous membrane. These areas may be the seat of the so-called Bednar's aphthæ. Midway in the raphe of the hard palate in the majority of newborn infants are seen one or two, at most three, yellowish-white, sago-like bodies. They are called Epstein pearls, and were first described by Epstein as collections of epithelial cells, the remains of embryonal life. They may be easily injured, and then become the seat of ulceration.

At the side of the hard palate over the alveolar process, above and below, the mucous membrane is thin and white in reflex. A slight traumatism may cause ulceration in this region. The tonsils of the newly born infant are hardly visible. The posterior pharyngeal wall has a glossy, smooth, bluish-pink reflex.

On close examination of the anterior pillars of the fauces of infants, bodies resembling drops of dew or vesicles are seen just in front of the location of the tonsil. These are collections of lymphoid tissue and are normal in the infant's mouth. They sometimes become inflamed and form aphthous ulcerations, and are then called herpes of the tonsil. There are also visible on the soft palate of children minute, miliary, transparent bodies having the appearance of vesicles. These are present in the normal state, and are likely to enlarge in diseases affecting the throat or in the exanthemata. They are aggregations of lymphoid tissue.

#### DENTITION.

The eruption of the temporary or milk-teeth begins about the sixth or seventh month, with the lower incisors, and ends about the third year, with the posterior molars. The eruption of the teeth, even in normal infants, varies within wide limits. The following table includes the most important facts concerning the normal eruption of the milk-teeth :

Two lower incisors . . . . .	{ 6th-10th month. Average 7th month.
Two upper lateral incisors . . . . .	10th-16th month.
Two lower lateral incisors . . . . .	12th-16th "
Four anterior molars . . . . .	14th-21st "
Four canines . . . . .	16th-25th "
Two upper incisors . . . . .	9th-10th-16th month.
Four posterior molars . . . . .	18th-36th month.

The second dentition begins in the seventh year, with the eruption of the first molar behind the second temporary molar. The central incisors appear about the eighth year; the lateral incisors, at the ninth year; and the last molars, from the eighteenth to the twentieth year, or later.

At the twelfth month a baby should have the upper and lower central incisors, with the two upper lateral incisors coming. The lower incisors may not appear until the eighth or ninth month, and may then be followed rapidly by others. I have seen several infants with one or two incisors at birth. These were imperfectly formed and resembled canines. As a rule prematurely erupted teeth should be extracted, as they lacerate the nipple in nursing. I have seen rachitic, bottle-fed infants who did not cut their first tooth until the twenty-fourth month.

**Rachitis** is a common cause of delayed dentition. Artificially fed infants are backward in cutting their first incisors. It is common to see bottle-fed infants cutting the lower anterior incisors at the ninth month. The infants may be in other respects normal. Rachitis affects the teeth of the first dentition mostly, but may influence the form and structure of the teeth of the second dentition. The teeth of the first dentition in rachitis are easily broken and are unnaturally white. In many cases the anterior incisors show an incurvation on the lower cutting edge, which is often mistaken by the inexperienced for Hutchinson's deformity. The first teeth in rachitis are easily eroded. It is not uncommon to see a rachitic infant with its whole dental system in process of decay. The permanent teeth present abnormalities in inordinate size and longitudinal furrows.

**Syphilis.**—The permanent teeth are affected by syphilis in a characteristic fashion.

**Hutchinson's teeth** are so called because they were first described by Jonathan Hutchinson. They are the only teeth of the permanent set which are pathognomonic of congenital or very early acquired syphilis (infancy) (Fig. 80). In a large experience with syphilis in infancy and childhood I have seen but few perfect examples of these teeth. The teeth presenting the deformity are the central upper incisors of the permanent set, and these only. "These teeth show a central single, rather broad notch." In this notch the dentine, lightly covered by enamel, is exposed. It is seen as a ridge in the



incurvation. The teeth are shorter and broader than is natural, and almost always have their angles sloped off. They are thus narrower at their cutting edge than higher up. They are seldom or never of good color, and frequently are not placed quite straight,

FIG. 80.



Hutchinson's teeth in a boy, twelve years of age.

but slope either toward or away from each other. Teeth which are the seat of erosion may resemble Hutchinson's teeth (Fig. 81). Fournier has described teeth in the temporary set which closely

FIG. 81.



Permanent teeth deformed through stomatitis in early childhood, resembling Hutchinson's teeth. Female child, nine years of age.

resembled Hutchinson's teeth. I have met an exquisite example of such teeth in an infant sixteen months old, the subject of syphilis (Fig. 82).

In syphilitic subjects we find the following deformities in the

permanent teeth.

These peculiarities are not characteristic of syphilis alone, but are found in those who are not syphilitic, but have suffered from stomatitis or dyscrasia of some kind. The changes are bilateral and symmetrical.

FIG. 82.



Central upper incisors of the first dentition resembling Hutchinson's teeth. Syphilis of the flat and long bones. Child, sixteen months of age.

**Dental Erosions.**—The most important erosions, such as those of Hutchinson just described, affect the central incisors. Other erosions give the teeth an incurvated appearance on their cutting edge. In this incurvation is seen a supernumerary crown

ribbed in a longitudinal direction (Figs. 83 and 84). The whole may be mistaken for Hutchinson's deformity. They result from malnutrition or stomatitis with faulty formation of dentine and enamel deposit in the eruptive period of the permanent teeth. The first molars show very characteristic deformities, which Fournier

FIG. 83.



Upper central incisors, with erosions not syphilitic.

FIG. 84.



Lower incisors, with erosions not syphilitic. Child, eight years of age.

places next in importance to those of the Hutchinson teeth, but does not regard as pathognomonic of syphilis, although they are met in syphilitic subjects. This deformity of the first molars is shown in Fig. 85, taken from a child who showed other erosions, but gave no history of syphilis. I have seen these erosions very well marked in children who had positive syphilitic manifestations. The top of the crown is constricted, and there appears to be a double crown. Erosions are also seen in the canine teeth.

**Microdontism.**—The teeth are quite small, but if cared for remain perfect in shape, pearly and transparent. They are seen in children whose parents may have suffered from syphilis. The children may also have obstinate eczema of the anus (parasymphilitic). Microdontism may occur also as a result of any non-syphilitic dyscrasia.

**Dental infantilism**, described by Fournier, occurs in children who are syphilitic. Small teeth presenting erosions are interspersed among teeth which are normal in size and shape.



**Amorphism**, or the tendency of a tooth, such as the incisor, to take the shape of a canine, has been noted by Fournier. I have also met with cases of this deformity in congenitally syphilitic children. It is seen in children who have had syphilis, but may be met with in those who have no such history.

Children, subjects of syphilis, do not always present deformities of the teeth. In a girl of fourteen years, who gave a history of infantile syphilis, and who had late manifestations, such as gummata

FIG. 85.



Erosion of molars, not necessarily syphilitic.

FIG. 86.



Molar tooth, showing erosion at crown. Boy, twelve years of age; same patient as with Hutchinson's teeth.

in almost all the bones, joint-affections, and gummata of the liver, the teeth, both upper and lower, were normal, of great beauty, and well preserved.

### Pathology of Dentition.

The period of infantile dentition is one of great physiological activity and growth. The organism is forming at this time. The nervous system is in a condition of instability. The gut is exposed to all varieties of infection, and is very susceptible to them. During this period the infant or child suffers from a number of diseases and exhibits a variety of symptoms which in former times were difficult of interpretation. With advancing knowledge and the possibility of making more accurate diagnoses than were formerly feasible, the diseases incidental to dentition have become more a matter of speculation. There are clinicians of note who still believe that irritation of the trigeminal branches by an erupting tooth may cause reflex eclampsia. It is difficult, and not necessary, to pass here on the status of that section of infantile pathology which treats of the disorders incident to dentition. In the presence of mystifying symptoms the physician should make a very careful examination, in order to make the diagnosis. Clinical observation of a case for a few days, and accurate registration of the pulse, respiration, and temperature every three hours, may show that the diagnosis of dentition must give way to something more tangible.

**Should the Gums be Incised?**—I have often found the tooth-sacs to be swollen and the seat of painful distention just before the eruption of the teeth. In one case the tooth-sac was distended by a

hemorrhage into its cavity. Under these conditions I have never yielded to the entreaties of the mother to lance the gums. I have seen no ill effects result from this *laissez faire* method. Very painful ulcerations result from friction, and uncontrollable hemorrhage may follow incision. In cases in which the sacs are distended, the functions of the stomach and gut should be kept normal, in order that complications may not be added to existing conditions. In rare cases I have seen suppuration in the tooth-sac, and have incised. In some cases of scurvy the tooth-sacs are distended and bluish in appearance. Treatment of the scurvy improves this condition.

### APHTHOUS STOMATITIS.

(*Stomatitis Aphthosa.*)

In this condition there are formed on the soft and the hard palate, the mucous membrane of the gums and tongue, and on the inner surface of the lips and cheeks, small round yellowish superficial ulcerations. These ulcerations, which vary in form and number, may coalesce and form irregular plaques. It is a question whether the ulcerations are the remains of vesicles which have burst, thus exposing an ulcerated base, or whether they are primarily ulcers. I am inclined to the former view, for in the so-called herpetic aphthæ of the tonsils the natural development of the aphthous ulcerations can be observed to advance from the vesicular to the ulcerative stage. This condition is very common in infancy and childhood, and according to Monti is most frequent between the first and the third year.

The **etiology** is still obscure. Some authors consider aphthous stomatitis an acute infection derived from the gut, possibly caused by toxins generated in contaminated milk (Forcheimer, Ritter, Kmeriem, Schamtyr). Others, basing their opinion on bacteriological studies, regard it as a purely local affection. The clinical course of the disease tends to support the former view. It has been compared by Forcheimer and others to the so-called foot-and-mouth disease of cattle.

The condition may occur idiopathically or may complicate intestinal infection, the exanthemata, bronchitis, tonsillitis, and pneumonia. Some authors believe that the affection may be communicated to others by the secretions of the mouth.

**Bacteriology.**—The forms of bacteria most commonly found in the ulcerations are the various streptococci and staphylococci (Judasohn). Bernabei has found the pneumobacillus of Friedländer. As these bacteria are present in the normal secretions of the mouth, it is doubtful whether they bear a causal relation to the condition.

**Symptoms.**—These aphthæ vary from the size of a pin's head to



that of a split pea. They are invariably surrounded by an areola of inflamed mucous membrane. The outline of the ulceration may be round or irregular; as a rule the ulcerations are superficial. At the line of junction of the teeth and gums they may show a tendency to bleed if touched. There is considerable pain, with salivation, and in young infants also a distinct febrile condition and green diarrhoeal movements. In other cases there may be an accompanying angina with swelling not only of the lymph-nodes at the angle of the jaw, but also of those underneath the jaw. In addition there are loss of appetite, and restlessness at night.

**Course.**—In well-nourished infants and children the tendency is to limitation of the aphthæ and spontaneous recovery within three or four days. In marantic or badly nourished children in unhygienic surroundings, the aphthæ are likely to spread, the ulcerations presenting the appearance of a mixed infection. Such cases are difficult to control. As a rule, however, the disease runs its course without leaving any lasting ill results.

The **treatment** of the cases in which the ulcerations or aphthæ remain discrete and in which mixed infection does not occur is begun with a saline cathartic, such as magnesia, or a dose of calomel. The mouth should not be washed. Careless attempts to cleanse the mouth are likely to cause the aphthæ to coalesce and spread, and also to cause intense pain. I administer a small dose of ferric chloride, made up with glycerin, every three hours. In most cases this will suffice. The use of potassium chlorate should be avoided with infants. If the edges of the gums adjacent to the teeth are affected, the teeth should be gently washed three times daily with a weak solution of tincture of myrrh or a saturated solution of boric acid. If the aphthæ coalesce, they should be touched once daily with a 2 per cent. solution of silver nitrate. With intractable young children, care should be taken in washing the mouth not to traumatize the unaffected mucous membrane.

### Bednar's Aphthæ.

Bednar's aphthæ, named after the distinguished Viennese pediatricist who first described them, are two symmetrical ulcerations over the hamular process of the palate bone, seen in the newly born or very young infant (Fig. 87). In a large number of cases they are the result of traumatism. The finger of the nurse impinges on the processes of the palate bone when it is introduced into the mouth, and abrades the epithelium. Any bacteria which may be present in the mouth or on the finger thus gain foothold and ulceration results. Epstein has shown that in the newly born infant such ulcers may be the starting-point of a general sepsis.

The infant may refuse to nurse, or if it does attempt to do so, the pain caused by the act of suckling causes it to desist. There may be intestinal disturbance, manifested by greenish stools and caused by infection of the gut by the bacterial flora of the ulcerations.

FIG. 87.



View of hard and soft palate. Lateral ulcerations—so-called Bednar's aphthæ. The central dark spots in the raphe are Epstein's pearls.

**Treatment.**—The ulcer should neither be washed nor traumatized. The rest of the mouth and tongue should be washed gently twice daily with a saturated aqueous solution of boric acid. The ulcers should be touched once or twice a day with a 10 per cent. solution of silver nitrate applied with a small piece of cotton on an applicator.

### SPRUE.

(*Thrush*; *Muguet* (Fr.); *Soor* (Ger.).)

Sprue is a parasitic growth on the mucous membrane of the buccal cavity of the newborn or very young infant. It may spread to the nose in cases of cleft palate; in other cases it may spread to the pharynx, larynx, œsophagus (Parrot), and even to the stomach (Parrot, Henoeh, Northrup). The latter situation is not favorable to its growth. The parasite has been found in the movements of infants suffering from the disease.

**Nature.**—Sprue is one of the mould fungi. Its classification by various authors varies with the species examined. Older authors classed sprue with the oïdium as *Oïdium albicans*. Rees, Grawitz, and Kehrer classified it as a *Mycoderma albicans*, consisting of conidia and mycelia. Plaut classifies it as a common mould fungus (*Monilia candida*).



In the early stages it presents large or small irregular whitish masses. These may at first be very minute, covering only the summits of the papillæ of the tongue. On the buccal mucous membrane they may be as large as a pin's head or coalesce into masses resembling curdled milk. They may be seen on the roof of the mouth, on the soft palate, tonsils, and posterior pharyngeal wall. If the affection is progressive, the tongue and inner surface of the cheeks become coated with a white closely adherent pellicle. In neglected cases the sprue may be of a yellowish color if *sarcinæ* are present, or blackish or grayish in hue if other fungi have obtained lodgement. Considerable force is required to dislodge the growth from the mucous membrane, and the operation will cause bleeding and considerable pain and traumatism.

**Occurrence.**—The organism is introduced into the mouth from without. It is present in the vaginal secretions of the mother, and has been found on the breast nipple. An abrasion of the mucous membrane must exist in order that the fungus may obtain lodgement. It is therefore found in infants whose mouths have been harshly washed with unclean fingers or into whose mouths unclean breast or bottle nipples have been introduced after harsh washing. The fungus having gained access to the cement-substance between the epithelial cells, proliferates into the deeper layers of epithelium, and may even invade the underlying connective tissue. Sprue carries with it any other bacterial flora which may be present in the mouth. A perfectly normal mucous membrane is not vulnerable to sprue. The sprue conidia and mycelia are found in the secretions of the mouth of the normal baby. Sprue is seen chiefly in infants whose health is below the average, who are inmates of institutions, or who have been in unhygienic surroundings.

**Symptoms.**—The local symptoms are due to the presence of the growth. In mild cases the patches are few in number and very minute. In neglected cases not only is the whole mouth the seat of the disease, but also evidences of infections of a pyogenic nature occur in the form of erosions of the buccal mucous membrane, yellowish plaque-like ulcerations and fissures which bleed easily. There is also dryness of the mucous membrane which has not been attacked or which has been freed from the fungus. Sprue, in fact, causes distinct reaction of the healthy mucous membrane in the vicinity of its invasion. Infants, even in the early stages, suffer from mild disturbances of the gastro-enteric tract, manifested by vomiting and greenish movements. In neglected cases marantic symptoms are also present. Older writers (Parrot) believed sprue to be a causal factor in athrepsia. It is simply regarded as a complication.

That pain is felt is evinced by the lack of desire to nurse the

breast. A febrile movement occurs if the intestinal tract is involved.

**Treatment.—Prophylactic.**—Everything that is introduced into the mouth of the infant should be scrupulously clean. If the infant is breast-fed, the breast nipple should be cleansed before and after nursing with a pledget of cotton moistened with boric acid. The infant's mouth should not be cleansed after nursing. In cases in which the roof of the mouth has been carelessly cleansed there are not only the aphthæ of Bednar, but also sprue and other aphthæ in the median line as a result of traumatism to Epstein's pearls. If infants are fed artificially, the nipple of the nursing-bottle should be boiled in soda solution once. (See page 46.) If these precautions are carefully observed, and unclean fingers never introduced into the infant's mouth, sprue will rarely if ever occur. The normal epithelium and normal secretions are safeguards against the fungus.

**Curative.**—The growth should be removed by cleansing the mouth gently three times a day with a saturated solution of boric acid. The utmost gentleness should be used. Even in mild cases the removal of the sprue may extend over a number of days, because the parasite quickly reproduces itself. I use one piece of absorbent cotton for the roof of the mouth, another for the tongue, and another for the cheeks and lips. If it can be avoided, the mucous membrane should not be caused to bleed. If aphthæ exist, they should be touched lightly with a 2 per cent. solution of silver nitrate. The bowels should be opened by an initiative mild cathartic. Everything should be scrupulously clean. The severe cases, in which there is a septic condition due to extension of the sprue to the gastro-enteric tract, occur chiefly in foundling asylums. The infants die of septic infections. In private practice the prognosis is good if the case is seen early and correctly treated. Baginsky recommends potassium permanganate (1:150); others recommend corrosive sublimate (1:2000), but boric acid will be found to be equally satisfactory.

Henoch describes cases of sprue of the stomach. This is admittedly rare, and occurs in the form of slightly prominent plaques. Parrot describes sprue of the gastric mucous membrane as not infrequent.

### TOXIC STOMATITIS.

I have seen a number of cases of stomatitis caused by irritant poisons, such as potash and ammonia. The children so affected had attempted to drink a solution of potash or ammonia from a bottle left within their reach.

The **symptoms** were purely local. The mucous membranes of



the lips had a characteristic œdematous, swollen, and transparent appearance, the buccal mucous membrane and the tongue were pale and œdematous, and the papillæ were erect and transparent.

The **treatment** was expectant. A mixture containing bismuth subcarbonate seemed to give most relief. On subsidence of the œdema the mucous membrane presented a dry appearance. Sometimes small aphthous ulcerations appeared, which healed under applications of a 2 per cent. solution of silver nitrate.

In one case, five years of age, symptoms of œsophageal stricture were present three months after the ingestion of the irritant. Strictures of the œsophagus are more common after the ingestion of potash or lye solutions than after corrosion by ammonia.

### ULCERATIVE STOMATITIS.

(*Stomatitis ulcerosa*; *Stomatocœc*; Ger., *Mundfäule*.)

Ulcerative stomatitis is a disease of the mucous membrane of the mouth, gums, and tongue, which is characterized by ulceration with a fetid odor.

The **etiology** is still obscure. Frühwald and Bernheim found bacilli and spirochætæ (spirilla) in the ulcers. The fetid odor of the breath was reproduced in the cultures of Bernheim. The bacillus is lanceolate in form and resembles the diphtheria bacillus. These bacilli and spirilla are probably identical with those described in 1896 by Vincent as occurring in hospital gangrene.

The affection is most common between the fourth and the eighth year. The period of infancy seems to be exempt, in my opinion because of the absence of teeth. It occurs in children who have been neglected or who have lived in unhygienic surroundings, and is therefore very common in clinics and dispensaries. In the milder forms there is a line of yellowish ulceration along the margin of the gums at the point of contact with the teeth, and the adjacent mucous membrane is red and inflamed. When the gums are touched either in washing or in examination, bleeding occurs. There is a fetid odor of the breath, the tongue is coated; some children have pain and loss of appetite, and a slight febrile reaction. In the severer cases there are deep ulcerations along the margins of the gums, which bleed on the slightest provocation. Ulcers with a greenish-yellowish base are seen along the border of the tongue and beneath it. In these cases the lymph-nodes beneath the body of the jaw are enlarged and painful as a result of the infection. The salivation, pain, and local disturbance are considerable, and the fetor is marked. The buccal mucous membrane at the points of contact with the teeth may be deeply ulcerated, indurations of the tissues of the adjacent mucous membrane being also present. Small particles of

secretory tissue are seen to flow away in the saliva. So great is the pain that some children refuse to open the mouth or partake of food. I have seen the teeth become loose and necrosis of the alveolar process occur in places. Under the latter condition there is much swelling of the tissues above and beneath the jaw (lymph-nodes). The tonsils may also be the seat of ulceration of the same character as that occurring at the lateral margin of the tongue.

**Treatment.**—Cleanliness is the first step toward lessening the intensity of the inflammation. The mouth is washed every three hours with a solution of potassium chlorate, made by adding a teaspoonful of the saturated solution to a small glassful of water, or with a 0.5 per cent. solution of formalin. Internally, liberal doses of ferric chloride, made up with glycerin and water, have given the best results. If there are extensive ulcerative processes along the gums, the line of ulceration is gently touched once a day with a 10 per cent. solution of silver nitrate. In addition, the patient must have an abundance of fresh air, and is given a nutritious fluid diet, with fresh fruits and a small allowance of wine.

### PSEUDODIPHTHERITIC STOMATITIS.

This form of stomatitis was first accurately described by Epstein. It is seen in newborn infants who have sustained a traumatism of the mucous membrane of the mouth. An infection of the injured membrane with streptococci results in the formation of a membrane resembling that seen in true diphtheria. These cases occur in foundling-hospitals and amid unhygienic surroundings. The pseudomembrane is of a greenish-yellow hue, and may spread over the hard and the soft palate, the tongue, and the pharynx. It may involve secondarily the entrance to the larynx, as happened in the cases of Epstein, and the epiglottis and œsophagus as well. Gastro-intestinal symptoms and secondary septic pneumonia are developed. The temperature may, as in other cases of sepsis, be normal, or even subnormal. As a rule, the lymph-nodes are not enlarged. The condition must be differentiated from sprue and aphthous stomatitis. Aphthous stomatitis does not show any pseudomembrane; microscopic examination will aid in differentiating this disease from sprue.

**Treatment.**—Inasmuch as these cases are of septic origin, their course is progressive. On the other hand, small patches of membrane may be limited by applications of a 10 per cent. solution of silver nitrate. The membrane should not be peeled off, nor should the mouth be washed out with the finger. Antistreptococcic serum is of no use in these cases.



### GONORRHOËAL INFECTION OF THE MOUTH.

This affection, sometimes called gonorrhœal or blennorrhœal stomatitis, is an infection of the mucous membrane of the mouth by the gonococcus of Neisser. Infection occurs only in places where the mucous membrane has been injured. There may be an associated gonorrhœal infection of the eyes or the vulva and vagina. The infection may be introduced into the mouth by the fingers of the nurse or mother. If the mother is suffering from gonorrhœa, infection may occur at the time of birth or subsequent to parturition. The cases thus far reported (Rosinski, Kast) have developed from two to thirteen days after birth.

**Symptoms.**—The constitutional disturbance is slight; there is no fever, no pain, and no interference with suckling. The lesions occur on those parts of the hard palate most likely to suffer from traumatism and subsequent infection—the parts favored by Bednar's aphthæ, the median raphe in the alveolar processes of the hard palate, and the anterior two-thirds of the tongue. Inspection reveals yellowish-white patches, due to infiltration of the superficial epithelial layers of the mucous membrane with inflammatory products. There is no pseudomembranous formation, but a pultaceous thickening. There is little tendency to spread, and no inflammatory reaction of the adjacent mucous membrane. The discharge is so slight that the saliva remains clear.

Examination of the secretion from the patches on the hard palate (which are generally symmetrical) and on the tongue reveals the presence of abundant gonococci not only on the surface, but also invading the mucous membrane along the cement-substance between the epithelial cells. The infection differs from that seen in adults (Cutler), in whom great constitutional disturbance and severe inflammation of the whole mucous membrane of the mouth are combined with a profuse ichorous buccal discharge and with pain. The tendency is toward rapid recovery.

The **treatment** is limited to the enforcement of strict cleanliness, and to local applications of weak solutions of silver nitrate (2 per cent.). The mouth may be washed twice daily with a solution of protargol.

### NOMA.

(*Cancerum Oris.*)

Noma is a specific bacterial affection which attacks the tissues of one or both sides of the face, resulting in gangrene and destruction of the soft and hard parts. Babes and Zambolovici differentiate it from all other forms of gangrenous stomatitis and gangrene, such as those described by Henoch as occurring on the vulva.

The **etiology** is still obscure. Investigations thus far tend to show that several conditions clinically similar have been found to have a diverse etiology. Babes and Zambolovici isolated a very minute bacillus, and by inoculation experiments in animals produced typical noma. They found that this bacillus extends through the mucous membrane of the mouth, especially that of the gums. Accompanying it are a large number of streptococci, spirochaetae, and other bacilli. The latter play an active secondary rôle in the production of the gangrene. Gangrene is caused by an overwhelming bacterial invasion of the tissues. The toxins produced cause death of cell-life and necrosis in mass. In another set of cases, Walsh found the bacillus of diphtheria. These cases would appear to correspond to those published by Freimuth and Petruschky, who found a bacillus identical with the diphtheria bacillus in cases of noma of the vulva.

The greater number of cases of noma occur after measles. It may follow any of the exanthemata, typhus, typhoid fever, or any disease through which the power of resistance to infection is lessened.

**Symptoms.**—Henoch and Baginsky hold that in many cases an ulcerative stomatitis has preceded the main affection. The disease begins on the mucous membrane and invades the cheeks from within. Henoch alone has seen it begin from without in the form of a phlegmon of the cheek. It is first seen as a small ulcer with a blackish-gray base on the buccal mucous membrane opposite the teeth, or it may begin as a vesicle with serosanguinolent contents. After a period of time varying from a few hours to three or seven days the tissues of the cheeks become brawny and œdematous, the œdema involving the eyelids and lips. A dark, livid area finally appears on the corresponding exterior surface of the cheek. This area becomes black and gangrenous. Perforation and spreading of the gangrene rapidly result. The jaw may necrose and the teeth fall out. The process may spread downward along the neck, involving the shoulder in an œdematous, emphysematous, gangrenous mass. The indurations of the tissues of the cheek occurring in many forms of stomatitis ulcerosa should not be confounded with this affection; in these forms of induration gangrene is absent. In all cases of noma a marked gangrenous odor pervades the atmosphere about the patient.

The general condition of many cases is astonishingly good at first. The children seem unconcerned, and sit up in bed and play. The organism finally succumbs to the toxæmia accompanying such extreme destruction of tissue. There may be a febrile movement ( $103^{\circ}$  to  $104^{\circ}$  F.,  $39.4^{\circ}$  to  $40^{\circ}$  C.). The swallowing of gangrenous products in some cases causes an intensely prostrating and uncontrollable diarrhoea of a septic character. There is little or no



pain. Death results within two or three weeks, either through general toxæmia and heart failure or complicating pneumonia.

**Occurrence and Prognosis.**—From a study of the literature, noma is found to occur most frequently between the second and the seventh year. The mortality is very high—fully 75 per cent. (Woronichin).

**Treatment.**—The most diverse methods have been employed in an endeavor to arrest the progress of the affection. To support the strength of the patient is the first consideration; careful ventilation, antiseptic and deodorizing solutions to mask the gangrenous odor, good food, and wine, are all of service.

The local treatment varies. Some authors advise dusting iodoform on the gangrenous area; others advocate the use of caustic zinc pastes in order to determine the line of demarcation between the gangrenous and healthy tissues. The Paquelin cautery with knife-blade attachment has been employed to remove the gangrenous tissue. Solutions of boric acid, thymol, and salicylic acid, should be freely employed to keep the mouth and parts clean.

In those cases, probably a distinct set, in which the bacillus of diphtheria is found, the antitoxic diphtheria serum should be injected in proper doses.

## THE TONGUE.

### Congenital Anomalies of Size.

(*Macroglossia.*)

The tongue of some infants who are otherwise normal is unusually large and protrudes slightly from the mouth, but is of normal shape. It is pointed, but somewhat thickened in the middle (Fig. 88). As the infant grows older this anomaly becomes less apparent. In extreme cases the tongue protrudes from the mouth as a tumor mass. It is discolored—generally of a livid hue—and becomes ulcerated, especially at the line of the teeth. Infants thus affected cannot nurse, and the tongue must be reduced in size by surgical means. This congenital enlargement of the tongue may be due to an increase either in the connective or muscular tissues, or in both. In other cases the lymph-spaces of part or the whole of the organ are dilated—there is a lymphangioma of the tongue.

In cretins and the Mongolian forms of idiocy the tongue is also enlarged. It is broad, thick, and flat, and protrudes from between the lips.

### Ringworm of the Tongue.

(*Wandering Rash of the Tongue; Lingua Geographica.*)

Ringworm of the tongue is a common affection of infants and children. It was probably first described by Santulus in 1854.

Parrot regarded it as a symptom of hereditary syphilis—a view which has no clinical support.

In 103 cases reported by Böhm, the condition occurred sometimes in early infancy, sometimes as late as the twelfth year of life, and was most frequent between the first and the second year.

FIG. 88.



Simple macroglossia. Infant, twelve months of age.

The **etiology** is obscure. Böhm believes it to be connected with a lymphatic diathesis (scrofulosis). It is found chiefly among children of the lower classes. It may, however, be seen in children in good hygienic surroundings and who are otherwise healthy.

If scrapings from the borders of the patches of an affected tongue be examined microscopically when fresh, large numbers of zoöglæa of coccus form, in some cases mingled with sarcinæ, will be seen. The presence of the latter micro-organism explains the yellow color of the border of the patches in some cases. The disease sometimes affects several children of a family.

The **symptoms** are limited to the appearance of the patches on the tongue. At the tip, but most frequently at the sides of the tongue, are seen areas sharply circumscribed by narrow, sinuous, perfectly oval or round borders (Fig. 89). The border is not only distinctly raised above the epithelium of the tongue, but also is of limited breadth and has a more pronounced whitish or yellow-white



color than the rest of the tongue. Inside this border, if the patch is oval, the tongue seems to be denuded of its epithelium and is reddish in color. This condition should be differentiated from desquamation of the epithelium on the dorsum of the tongue, which presents a similar appearance, but in which the patches have not the band-like border. Children do not appear to suffer inconvenience from this condition of the tongue.

**Treatment** of the most diverse kinds, including local application of tincture of iodine and the use of ferric chloride, has in my experience failed to produce results.

FIG. 89.



Ringworm or wandering rash of the tongue, *lingua geographica*.

FIG. 90.



Epithelial desquamation of the tongue.

### Desquamation of the Epithelium of the Tongue.

In this condition, which has been confounded with that just described, there are seen areas of irregular size and apparently denuded of epithelium. The boundary of these areas is sharply outlined, but the epithelium bounding the areas is apparently normal (Fig. 90). The tongue looks as if the epithelium had been scraped off. The condition demands no treatment, since it is only a symptom of mild derangement of the digestive processes.

### Tongue-swallowing.

Tongue-swallowing is a term applied to a peculiar phenomenon seen in some infants who are the subjects of nasal obstruction. Infants normally breathe through the nose when at rest, the tongue being in contact with the roof of the mouth. If nasal breathing is obstructed either by swelling of the mucous membrane or by deformity of bone, the infant experiences great difficulty in breathing

through the nose. As a result, not being accustomed to keeping the mouth open and the tongue on the floor of the mouth, the ineffectual efforts at nasal and mouth-breathing cause the infant to draw the tongue inward. The tip of the organ folds on itself, and may be drawn backward into the mouth in the efforts at mouth-breathing, causing a peculiar snapping noise to be heard on inspiration.

**Treatment.**—The remedy in these cases is nasal douching, and dilatation of the nasal passages with pledgets of cotton. The cotton is rolled around a probe or applicator, moistened with castor oil, introduced once a day into the nares, and allowed to remain about five minutes.

### **Tongue-tie.**

Tongue-tie is a condition for the relief of which the physician is frequently consulted. Some mothers will ascribe inefficient nursing to this condition. With a breast secreting sufficient milk tongue-tie would not prevent nursing. The existence of the condition is readily detected if the organ is bifid at its tip when protruded. The frenulum will in such cases be seen to extend to the extreme tip of the tongue in a fan-shaped manner.

**Treatment.**—The frenulum being membranous is easily divided. It should be caught in the bifid groove of the pocket-case director and made tense, and the membranous portion divided with a pair of round-ended scissors. The ends of the scissors should be directed to the floor of the mouth. There is little bleeding. The infant should be placed at the breast directly after the operation, so that the act of suckling may stop the hemorrhage.

### **MALFORMATIONS OF THE UVULA.**

The uvula is often bifid in infants. This condition is only of anatomical interest. There are cases in which the uvula is relaxed and elongated. In one case, in a boy five years of age, the uvula was so long that it gave rise to an incessant night-cough. On excision of the uvula the cough ceased.

### **ACUTE RETROPHARYNGEAL ABSCESS.**

*(Idiopathic Retropharyngeal Abscess; Retropharyngeal Lymphadenitis.)*

The retropharyngeal space, according to Gillette, is the seat of several lymph-nodes, which are intimately connected with the lymph-vessels and lymph-spaces of the tonsils, and also with the system of lymph-vessels of the soft palate, these being also connected with the deep lymph-nodes of the face and neck. Processes such as catarrhal angina, diphtheria, scarlet fever, measles, or any lesion of the mouth,



are likely to involve the retropharyngeal nodes (Karewski). Sometimes only the lymph-nodes in the median line of the retropharynx opposite the base of the tongue are affected. In this form the tumor in the midline is seen when the mouth is opened. In other cases several lymph-nodes are involved, and the process is then seen both as a swelling in the mouth and as an external swelling at the side of the neck.

The swelling appears at or beneath the angle of the jaw, in front of or behind the sterno-mastoid muscles. Retropharyngeal abscess may occur in the following forms :

1. Acute retropharyngeal abscess :
  - a. That which points wholly in the mouth.
  - b. That which points both externally and internally.
  - c. That which forms a tumor chiefly external.
2. Chronic tuberculous retropharyngeal abscess.
3. Septic retropharyngeal abscess.

This third class of retropharyngeal abscesses are those which complicate or follow the exanthemata, and which have a tendency to burrow downward, bursting into the mediastinum or to involve important structures, such as the large arteries in the neck, thus causing fatal hemorrhage. A few such cases occur in the literature (Bokai).

**Frequency and Etiology.**—Retropharyngeal abscess is peculiarly a disease of infancy and early childhood. The frequency diminishes in later childhood, the disease being rare after the fifth year. Of 77 of my cases, 4 occurred between the first to the third month ; 10 between the third and the sixth month ; 41 between the sixth and the twelfth month ; 19 between the first and the fifth year, and the remainder after the fifth year. One infant was only one month of age, and in two cases the patient was two months of age. The figures correspond to those of Bokai. The frequency in early infancy is probably explained by the structure of the retropharyngeal lymph-spaces and the susceptibility of the lymph-nodes to suppurative infections at that period of life.

Simon has described the lymphatics in the retropharyngeal region of infants and children as forming a small network of lymph-vessels and nodes on either side of the median line. This lymphatic network is situated between the superior constrictor and the aponeurosis of the prevertebral muscles. After the third year of life these lymphatics and nodes are said to disappear. This fact, as Blackader points out, would indicate a close connection between the time of activity of these nodes and the period when retropharyngeal abscess is most prevalent. It would help also to explain the absence of this form of abscess in older children and in adults who are frequently affected by tonsillar (quinsy) abscess.

I have examined the pus from many of these abscesses, and found

that it contains quite uniformly a streptococcus of the short or the long variety, not as a rule very virulent. It may be assumed that in all probability these bacteria are the essential cause of the abscesses. They gain access to the retropharynx either through the tonsils or the mucous membrane of the pharyngeal space. The abscess may thus be secondary to any form of inflammation of these structures. It occurs as a complication of simple tonsillitis, pharyngitis, influenza, or any of the exanthemata.

The **symptoms** of retropharyngeal abscess are not at first distinctive. The development of the abscess is insidious. At the outset there are the symptoms of ordinary tonsillitis or pharyngitis. The fever is high at the beginning. After the acute symptoms subside it is noticed that the lymph-nodes at the angle of the jaw continue to be enlarged, and that the fever continues to show a remittent type. There is some prostration, the infant does not nurse properly, cries, and is frequently restless. Inspection of the throat on the fourth or fifth day of a tonsillitis may reveal nothing except some swelling or œdema of the posterior pharyngeal wall or of the pillars of the fauces, no tumor being visible. After an interval of a few days, generally on the seventh or eighth after the initial symptoms, it is noticed that the voice of the infant has a nasal quality, that the head is thrown back, and that the breathing is noisy and nasal. Examination shows that the lymph-nodes at the angle of the jaw in front or behind the sterno-mastoid are swollen; inspection of the interior of the fauces shows a distinct swelling at the side of the pharynx pushing the tonsil and pillar of the fauces of that side forward. On introducing the finger a tense, fluctuating swelling, which may reach downward toward the larynx, can be felt. In other cases there is very little external swelling, and the internal tumor is situated nearer the median line, pushing the posterior pharyngeal wall forward. This swelling is covered by mucous membrane, is tense and fluctuating. If the tumor is allowed to increase in size, there is pronounced interference with the breathing. I have seen cases in rachitic infants in which the inspiratory sound was distinctly of a crowing character, showing incoördinate action of the vocal cords. These cases show great prostration and feebleness of pulse.

**Course.**—If not treated, the abscess may press on the larynx and cause asphyxia, or may burst spontaneously into the larynx, suffocating the patient if it occurs during sleep, or may burst into the ear through the Eustachian tube and discharge externally. All of these results are rare if the abscess is detected in time for incision.

The **diagnosis** of retropharyngeal abscess is difficult to the beginner, but is simple after the observation of one or two cases. The quality of the voice and the cry are so characteristic that after being once heard they are unmistakable. The breathing also is typical.



The external swelling is present in most cases, and the head slightly retracted. Finally, digital examination should always be resorted to in all cases in which a slight or marked internal swelling is present. The index finger of the right hand is passed into the mouth and the posterior pharyngeal wall palpated. If an abscess be present, it will be apparent as a hard or tense, globular, deep or superficially fluctuating tumor. Care should be taken not to mistake the prominence of the body of the seventh cervical vertebrae for an abscess. The bony tumor is deeper, as a rule, than the retropharyngeal abscess, and is not fluctuating. All manipulation should be carried out gently, else the abscess may burst and suffocate the patient or rude exploration may cause a peculiar form of collapse which sometimes follows digital examination in this region.

The **prognosis** of simple acute retropharyngeal abscess is good. Bokai lost only 4 per cent. of his cases. With early diagnosis and proper treatment recovery is the rule.

The **treatment** of acute retropharyngeal abscess is incision. This varies with the nature and location of the abscess. In the majority of cases the abscess is near the median line, and its wall is just beneath the surface of the mucous membrane. An internal incision will then afford immediate and permanent relief. In other cases the abscess is at one side and internal, and may also be safely incised from within. In making an internal incision the following method should be pursued: the child is wrapped in a blanket and held upright in the lap of the nurse, facing a good light. An assistant steadies the head from behind. The tongue is depressed with a tongue-depressor, and a bistoury, with the edge guarded by rubber plaster, leaving only a half inch of the tip exposed, is plunged into the most prominent part of the tumor. When the pus escapes, the incision is enlarged from above downward. The instrument should not be directed toward the side of the neck, for fear of wounding a vessel. As soon as the pus escapes freely the head of the infant is thrown forward and the pus allowed to drain into a basin, pressure being made from without, on the side of the neck. The internal incision should be made as rapidly and as gently as possible. I have seen death result within a few hours from aspiration of pus in a case in which an abscess burst as a consequence of rough digital exploration. If necessary, the incision may be enlarged with a dressing-forceps. In some cases the wound should be prevented from closing by introducing the forceps daily.

There is another class of cases in which the deep cervical glands at the side of the neck are involved and the abscess points partly internally and partly externally. In these cases it is unsafe to incise from within, nor is complete relief afforded by so doing. The abscess should be approached from without through a careful dissection by a skilled surgeon. The tuberculous abscess is due t

spinal caries, and is best opened and drained from without, as are also septic abscesses.

### RETRO-ŒSOPHAGEAL ABSCESS.

Retro-œsophageal abscess is a rare affection of infancy and childhood. Crozer Griffith has noted 15 cases in the literature. His own case was that of a child twenty-one months of age. The cause of the abscess was caries of the lower cervical and upper dorsal vertebræ.

The **symptoms** are obscure, and in the majority of cases no diagnosis was made. The principal symptom is a harassing cough persisting for several months, finally becoming croupy and brassy. In the final stage the dyspnoea causes marked diaphragmatic retraction of the lower part of the throat. There is no dysphagia and no such change in the voice as in retropharyngeal abscess. Inspection of the throat may reveal a slight external swelling over the cervical or dorsal vertebræ.

**Treatment.**—If abscess is diagnosed or suspected, an incision should be made along the œsophagus, and the abscess drained from the outside.

### ADENOID VEGETATIONS.

It is not within the scope of this work to give more than the diagnostic bearings of these growths as they occur in infants and children. On inspection, the posterior nasopharynx in the normal infant is frequently seen to be the seat of more or less adenoid tissue. The diseased condition is simply an exaggerated growth of the tissue which is normally present in this space. Clinically there are three distinct classes of cases that suffer from adenoids:

The first class comprises those in which the adenoids cause few or no symptoms. The children when in good health breathe through the nose and keep the mouth closed during sleep. They are peculiarly susceptible to slight colds or catarrh, and when thus affected the tonsils enlarge, the nose becomes obstructed by secretion, there is difficulty in breathing, and the patient sleeps with the mouth open. On the subsidence of the inflammatory condition the normal status is re-established. The children are subject to recurrent attacks of tonsillitis, and with each recurrence the symptoms of adenoids become more marked. The patients contract obstinate coughs which resist all treatment, and epistaxis occurs from causes apparently trivial.

The second class of cases comprises those in which, in addition to the enlarged tonsils, there are enlarged lymph-nodes in various regions of the body. The patients are pale and present all the



symptoms of lymphatism. Their voices have a nasal intonation, the lips are always parted, and they sleep with the mouth open (mouth-breathers).

The third class comprises the genuine cases of adenoids. The nasal passages are the seat of a chronic hypertrophic rhinitis, the tonsils are enlarged, there is obstructed breathing, and the mouth is always open. The infants and children make a peculiar snarling sound in breathing and have a stupid look. They are not necessarily lymphatic. Many children suffering from adenoids are slightly deaf, and all are subject to repeated catarrhal attacks.

Between the extremes are seen all gradations of the affection. Many children who suffer from adenoids are well developed and in other respects perfectly normal. The deformities of the chest which have been ascribed to adenoids can hardly be so regarded. They are coincidental. Many of them are due to rachitis in early life and to unhygienic living. To trace enuresis, chorea, and masturbation to the presence of adenoids, seems also somewhat extreme. Adenoids are an obstruction to the breathing, a menace to the hearing, and also a focus for repeated infections of the nasopharynx or the ears. These are sufficient reasons for their removal.

The **diagnosis** of the condition is made from the above symptoms, and also by digital exploration. Care should be taken that the finger used in exploring the posterior nasopharyngeal space is very clean and that the nail is smoothly trimmed. The parts should not be traumatized unnecessarily. The index finger passed up and behind the soft palate encounters soft masses of adenoid tissue which bleed easily and are readily crushed. They are sometimes pedunculated, and may be attached to the roof of the nasopharyngeal space or to the posterior portion of the nares. Some authors have advised the use of a shield in exploring this space. The skilful laryngologist prefers to use the mirror in examining these parts, and protests against the digital method.

The **treatment** of adenoid vegetations belongs to the special field of the nose and throat.

**Contraindications to Operations.**—The tonsils and adenoids being portals of infection, there are certain states in which operations in this region may be followed by reinfection. Thus cases of chorea with endocarditis, if still active, should not be subjected to operation. The chorea is likely to recur with greater severity, and the danger of a renewed heart lesion is great. Children who are in the active stages of endocarditis or recently recovered should not be operated upon. In all these cases palliative measures, such as sprays and douches, should be employed until the conditions above mentioned are thoroughly quiescent. In one case of chorea I saw an operation for adenoids followed in three days by a chill and high fever, endo-

pericarditis, chorea insaniens, and death within ten days. While such cases are exceptional, they teach the necessity of caution in deciding to operate upon the adenoids in chorea and heart cases.

### THE TONSILS.

The tonsils are really lymph-nodes, as has been shown by Stöhr and Hodenpyle. In severe forms of inflammation they are enlarged and the so-called crypts become plugged with bacteria and the products of inflammation (leucocytes, fibrin, serum). The crypts appear at the surface of the tonsil as yellowish specks. A catarrhally inflamed tonsil may not show them at the surface, because the products of inflammation do not coagulate, and are thus thrown off more readily. There is nothing specific about a lacunar or follicular amygdalitis. It is only a clinical picture of the large class of catarrhal inflammations, in all of which the crypts and the tissue of the tonsil are infiltrated with inflammatory products.

#### Acute Follicular Amygdalitis.

(*Acute Catarrhal Tonsillitis; Acute Lacunar Amygdalitis; Catarrhal Angina.*)

Acute follicular amygdalitis is an infectious disease, communicable either through the secretions or by direct contact, as in the act of kissing. It occurs both as a primary and as a secondary affection. As a primary affection, it occurs at all periods of infancy and childhood. It was formerly taught that follicular amygdalitis was rare in infants. This is scarcely true. Of 1284 cases of lacunar amygdalitis, 333 occurred in infants under the age of twelve months, and 76 from the first to the fifth month; of the latter, only 5 occurred in the first month. It is frequent in children from the second to the fourth year, but is more common after than before the fourth year. The tonsils are secondarily involved in the exanthemata—scarlet fever, measles, and varicella—and in parotitis, influenza, pneumonia, and pertussis. In all these affections they are red, swollen, and in some cases present the appearance seen in the typical lacunar type of the disease.

**Etiology.**—The predisposing causes of catarrhal tonsillitis or lacunar amygdalitis are exposure to cold, traumatism, and the swallowing of corrosive or irritant substances. The exciting causes of follicular or lacunar amygdalitis and catarrhal amygdalitis are the *Streptococcus pyogenes*, the *Staphylococcus pyogenes*, and the *pneumococcus*. The diplococcus described by Roux is also found in the tonsillar crypts.

**Symptoms.**—The affection rarely begins with a chill. The infant is restless, peevish, and wakeful at night; it breathes rapidly, and there are high fever and marked prostration. Nursing is in-



terfered with, not only on account of the pain in swallowing, but because in the majority of cases there is more or less rhinitis present. As a rule, the bowels are disturbed as a result of swallowing infectious secretions from the mouth with the food. The action of the bacteria in the gut is manifested in green stools, which are frequent and watery. Inspection of the throat should be conducted with patience and in a good light. The tonsils, normally very small, are seen to be enlarged and studded with whitish or yellowish-white points. The lymph-nodes at the angle of the jaw may be enlarged.

In older infants and children the tonsils are enlarged, and the crypts plugged with inflammatory products. The surface of the tonsils is covered with mucopurulent exudate, or there may be a small necrotic, ulcerated area in one of the tonsils. The neighboring structures, such as the uvula, the pharyngeal mucous membrane, the pillars of the fauces, and even the larynx, may share in the catarrhal inflammation. The lymph-nodes at the angle of the jaw may be enlarged. The fever, as a rule, is high at first, ranging from 104° to 105° F. (40° to 40.5° C.) or above. The pulse is correspondingly rapid, and the respirations may be increased in frequency.

The duration of a typical case of primary tonsillitis varies. As a rule, the temperature remains high for two or three days, with daily remissions. It then subsides and the patient convalesces. In some cases the temperature continues high for five or ten days, and then drops. In all of these cases there is some latent or apparent complication, such as retropharyngeal abscess, otitis, or, as has been recently pointed out by Packard and others, an insidious endocarditis. When otitis supervenes the tonsillar affection subsides. The fever, however, continues, with daily remissions. As a rule, infants and young children do not indicate the existence of pain in the ear. The patient is restless at night, and wakes with a start or in a peevish mood. In many cases the otitis can be diagnosed only by exclusion. In other cases the temperature continues high for a week or longer, reaching 103.5° F. (39.7° C.) during the day. The infant seems weaker, the tonsils are not enlarged or severely inflamed, the pulse is accelerated, and the respirations may number 40. In such cases the lungs show no sign of involvement, but careful examination of the heart will often reveal the presence of a systolic murmur at the apex and a slight increase of the area of cardiac dulness beyond the nipple. These are the so-called rheumatic cases. Frequently the urine shows a trace of albumin. In rare cases it contains in addition to the albumin elements pointing to parenchymatous irritation of the kidney. I saw a case recently, in a child six years of age, in which after a mild attack of tonsillitis there were a few casts, blood-cells, and a small amount of albumin in the urine.

Months elapsed before the urine ceased to show evidences of the nephritis. In these cases the albuminuria may assume the so-called cyclic character.

The **prognosis** of simple catarrhal tonsillitis is good, recovery taking place in a few days. On the other hand, tonsillitis is not the simple entity formerly supposed. In infants and children this is especially true. The physician should be watchful for possible complications and sequelæ, such as otitis, retropharyngeal abscess, endocarditis, and nephritis.

The **diagnosis** of tonsillitis is usually a simple matter. If an infant refuses the breast and the temperature is elevated, the throat should be carefully inspected. It is good practice to make a bacteriological culture with the secretions from the throat, even though the appearances are not diphtheritic at the first visit (for technique, see section on Diphtheria).

The **treatment** of acute tonsillitis is symptomatic. Sponging with cold water or water at 85° F. (29.4° C.) containing a dash of alcohol, will lower the temperature. A dose of quinine should be given twice daily, and if the lymph-nodes at the angle of the jaw are enlarged, cold applications should be made externally. Sprays are not required unless there is a harassing cough. Dobell's solution sprayed three times daily will relieve that symptom. In nursing infants the number of feedings by the breast or bottle is reduced. If there is disturbance of the bowel, a teaspoonful of castor oil or grain  $\frac{1}{2}$  (0.03) of calomel, given twice daily, will empty the bowel. The infant is then dieted on albumin-water or barley-water, or a solution of acorn cocoa or beef-juice and barley-water, until the intestinal irritation has disappeared. A return to a milk diet may be made as soon as the movements become normal. Small doses of ferric chloride have a beneficial effect on older children. In mixture form it is an excellent local application to the tonsils. The custom of giving potassium chlorate in this mixture is now generally abandoned, the drug being highly irritant to the kidneys. In nursing infants ferric chloride causes diarrhœa. For this reason it should not be administered to them for long periods.

### Herpes of the Tonsils.

Herpes of the tonsils are small vesicular formations seen on the anterior pillars of the fauces, just in front of the tonsils. They occur in a number of slight febrile conditions, may accompany an angina of a simple type, and are part of the clinical picture of aphthous stomatitis. The vesicles burst, leaving yellowish ulcerations of the size of a pin's head and surrounded by a pink areola. They heal without treatment after a few days.



## THE LARYNX.

### Acute Catarrhal Laryngitis.

(*Catarrhal Croup; Spasmodic Croup; Spasmodic Laryngitis; Pseudocroup.*)

**Etiology.**—Exposure to cold or wet are predisposing causes. Like the majority of catarrhal inflammations of the respiratory passages, acute catarrhal laryngitis is due to the invasion of bacteria. It occurs as a primary affection, and in a modified form is met with secondarily in measles and influenza. The classical form of "croup" is a primary affection, and is most common from the second to the fifth year. It is also seen in very young infants. One attack predisposes to others.

**Symptoms.**—Catarrhal croup or catarrhal laryngitis is an affection that causes much concern to mothers when a first attack develops without warning. During the day the infant may have had a mild coryza with a slight elevation of temperature. Toward evening a croupy cough, accompanied by croupy breathing or voice, suddenly develops. In some cases the symptoms remain mild, and only the cough disturbs the patients. They breathe freely, and dyspnoea is not marked. In other cases the infant or child goes to sleep free from alarming symptoms. Coryza may have been present unnoticed during the day. During the night the patient awakes with a croupy cough, which rapidly becomes worse. The breathing is noisy (croupy), and may be heard in an adjoining room. The cough is especially terrifying. The patients are restless, and cry during the paroxysms of coughing. In some cases they sit upright and gasp for breath. The face is pale and wet with cold perspiration. Fever may be slight or marked. In the majority of cases the dyspnoea is real; there is drawing inward of the suprasternal region and the peripneumonic groove at the epigastrium. Toward morning the dyspnoea, cough, and breathing subside, and the patients fall asleep, worn out with the night's suffering. The next day the patients are apparently well, with the exception of a slight cough, coryza, swollen tonsils, with redness of the pharynx. For two or three successive nights there may be a repetition of the attack. This condition should be differentiated from laryngismus stridulus. In the latter there is no fever, the breathing is stridulous during only a short spasmodic attack, and there is no croupy cough. On the other hand, pseudocroup may occur in children who are rachitic and the subjects of laryngismus. There are forms of diphtheritic laryngitis without the formation of membrane, which in their symptomatology are identical with the form of laryngitis above described. This is true in very young infants and in children above five years of age. A culture-test is the only certain mode of differentiating the affections. The pathological condition giving rise to pseudo-

croup is believed to be a swelling of the mucous membrane beneath the vocal cords.

**Treatment.**—The patient should be isolated. The crib should be placed under an improvised tent, and the tent filled with steam vapor saturated with benzoin or turpentine. Grains x (0.6) of calomel should be sublimed every two hours underneath the tent, as in diphtheritic laryngitis. This treatment is efficacious in the majority of cases, the croupy cough and breathing abating after the first inhalation. Turpeth mineral is a favorite remedy with many practitioners. An emetic is seldom necessary. I have never practised intubation in these cases. Although the dyspnœa was extreme, cyanosis was not present in the cases I have seen. Cases have been recorded in which resort was had to intubation to relieve dyspnœa and laryngeal obstruction. In such cases the suspicion is warrantable that there was diphtheritic invasion of the larynx.

#### LEADING AUTHORITIES REFERRED TO IN CHAPTER IV.

- Babes and Zambolovici*: Annales d'Institut. de Path., etc., Bucarest.  
*Bernheim*: Centralbl. f. Bakt., 1898.  
*Bishop and Ryan*: Jour. Amer. Med. Assoc., 1902.  
*Blackader*: "Retropharyngeal Abscess," Montreal Med. Jour., 1888-1889, vol. xvii.  
*Bokai, L., Jr.*: Paediatrische Arbeiten Henoch, 1890.  
*Crozer Griffith*: "Retro-œsophageal Abscess," Trans. Amer. Pediat. Soc., 1901.  
*Epstein*: "A Pseudodiphtherie," Jahrb. f. Kinderheilk., Bd. xxxix.  
*Forchheimer*: Archives of Pediatrics, 1892.  
*Freymuth and Petruschky*: Deutsch. med. Wochenschr., 1898, No. 38.  
*Frühwald*: Jahrb. f. Kinderheilk., Bd. xxix. p. 200.  
*Mayer, Emil*: "Affections of the Mouth and Throat," etc., Amer. Jour. Med. Sciences, 1902.  
*Nielot and Marotte*: Rev. de Médecine, 1901. (Vincent's bacillus.)  
*Rozinsky*: Zeitschr. f. Geb. u. Gyn., Bd. xii.  
*Walsh*: "Noma," Jour. Amer. Med. Assoc., 1902.  
*Sobel and Herrman*: N. Y. Med. Jour., 1901.  
*Vincent*: Annal. de l'Institut. Pasteur, 1896.  
*Woronichin*: Jahrb. f. Kinderheilk., Bd. xxvii.



## CHAPTER V.

### DISEASES OF THE GASTRO-ENTERIC TRACT.

#### PHYSIOLOGICAL AND ANATOMICAL FACTS CONCERNING DIGESTION IN INFANTS AND CHILDREN.

The reaction of the secretions of the mouth in the newly born infant, before it has partaken of food, is neutral or slightly alkaline (Ritter, Contaret, Korowin, Czerny). Little saliva is secreted. The gland secretion of the parotid contains ptyalin; that of the submaxillary gland possesses converting powers only after the second month (Montagne).

#### THE STOMACH.

The œsophagus enters the diaphragm at about the level of the ninth dorsal vertebra; the cardia is on a level with the tenth dorsal vertebra; the pylorus is in the majority of cases situated in the median line, but in some cases is slightly to the right of it. It is midway between the tip of the xiphoid cartilage and the umbilicus, and, being behind the liver, is not palpable. The stomach lies in an oblique position, passing from behind forward and downward. The pylorus is from two to two and one-half bodies of a vertebra lower than the cardia. In the newly born infant the inferior portion of the stomach has a fundus form (Pfandler), which later becomes more marked. Occasionally there is no fundus, and the stomach is then of cylindrical shape. Between the time of birth and the seventh month the fundus of the stomach increases to fully twice its original length (Pfandler).

The capacity of the stomach is still a matter of speculation. The absolute capacity, as given by Fleischman, Drenitz, Pfandler, Holt, and Rotch, varies with the method employed to determine it. The work thus far done has been carried out on the cadaver, and, moreover, the methods employed presuppose an amount of pressure (14 c.c. to 30 c.c.) of water which does not exist in the normal state during life. The stomach contracts after death (crystalline); the distention with air or fluids is thus partly artificial. Lastly, the stomach capacity is of little aid in determining the point at issue—the quantity of food which should be taken by a healthy infant at each feeding. Figures giving absolute stomach capacity are un-

ful only as indicating the actual size of the organ when full of fluid, a condition rarely present during life.

The following table is compiled from the sources mentioned. Pfaundler's results, which were obtained by careful computation, differ widely from those of others. They were obtained by post-mortem distention with fluid at a pressure of 30 c.c. of water. Fleischman distended the stomach at 14 c.c. of water pressure.

	FLEISCH- MAN c.c.	DREWITZ c.c.	PFAUND- LER. cc.	ROTCH. c.c.	HOLT. c.c.
At birth . . . . .	30	. .	30	30	36
One week . . . . .	45				
One month . . . . .	77	99	150	75	60
Two months . . . . .	79	115	175	96	99
Three months . . . . .	140	130	200	100	135
Four months . . . . .	. .	165	230	107	150
Five months . . . . .	290	253	266	108	170
Six months . . . . .	260	297	295	. .	264
Seven months . . . . .	. .	217	330		
Eight months . . . . .	. .	289	365		
Nine months . . . . .	. .	510	406		
Ten months . . . . .	375	350	445		
Eleven months . . . . .	. .	535	485	. .	243
Twelve months . . . . .	. .	500	515		
One to two years . . . . .	220	588	640		

**Function and Motility.**—The stomach of breast-fed infants empties itself in two hours after the ingestion of a full nursing. If the quantity of milk taken is small, a shorter time suffices. Bottle-fed infants taking cows' milk need fully three hours to accomplish the same result. This fact alone teaches that intervals of rest between the nursings, and a rest of four or five hours once in twenty-four hours, are necessary.

**Reaction.**—When digestion is not in progress the stomach contains a tenacious colorless mucus, neutral in reaction. When food is in the stomach, the reaction is acid.

Hydrochloric acid is normally present in the stomach of the infant (Leo, Van Puteren, Wohlman); lactic acid only occasionally. Heubner found 0.16 to 0.2 pro mille of lactic acid present. A considerable amount of hydrochloric acid unites with the salts and albumin of the milk, and is found as combined hydrochloric acid. When combination is no longer possible, the residue appears as free hydrochloric acid. The amount of free hydrochloric acid depends on the quantity of milk ingested, and varies from 0.8 to 2.1 pro mille. I have frequently failed to find it in the stomach contents of infants who are fed irregularly at frequent intervals. In healthy breast-fed infants free hydrochloric acid is found in from one and a quarter to two hours, and in bottle-fed infants in from two to two and a half hours after nursing. The effect of the lab-enzyme on the milk is marked in breast-fed as compared



with that in bottle-fed infants. In the former the action of the acid delays that of the lab-ferment fully an hour after feeding, while in the latter coagulation of the casein occurs at once and in large flocculi. The difference in the retarding action of the lab-ferment is due to the increased alkalescence of mother's milk, which requires more acid to neutralize the alkali, and thus to render coagulation possible: hence the greater digestibility of mother's milk and the rationale of adding lime-water to cows' milk for infant use.

Gastric contents containing free hydrochloric acid are antiseptic, while combined hydrochloric acid has no such properties.

In newly born infants lab-enzyme is present in minute quantities (Raudnitz); it is more abundant in older infants (Leo).

Pepsin is present in the stomach of the newly born infant (Hammarsten, Zweifel), as is also peptone (Leo, Van Puteren).

**Marking out the Stomach by Percussion.**—This procedure is difficult with infants and children. The normal stomach is rarely found outside of the left hypochondrium. The liver fully covers the stomach in the collapsed state. In the recumbent posture the stomach may be mapped out on the anterior abdominal parietes. It comes forward in the triangle formed on one side by the border of the left lobe of the liver and on the other by the border of the ribs. Above, the apex of the triangle is formed by a junction of the ribs and left lobe of the liver. Below, the base of the triangle is of variable length. In the axillary line the fundus in a moderately distended state is in contact with the thoracic walls, between the liver above and the spleen below. Above, it is separated from the lung resonance by a strip of dulness (the left lobe of the liver) which changes position with the movements of the diaphragm. The tympanic resonance reaches downward in a vertical direction from the sixth to the eighth rib. Behind this, tympany is limited by the posterior axillary line; in front, by the triangle above referred to. I have frequently been able to confirm these statements of Fleischman. Anteriorly, I have with the aid of a gastrodia-phane shown that the transverse colon passes in front of the stomach just beneath the liver. It should be remembered that tympanic resonance in the epigastrium is not always due to the stomach.

## INTESTINAL FERMENTS.

Little is known of the functions of the intestinal glandular apparatus. Many facts have been established as to the changes caused by the action of the secretions of the larger glandular organs on the food.

The **liver**, a very important organ in the newborn infant, is capable of forming glycogen.

The **gall-bladder** in the newborn infant is filled with a clear yellow secretion.

The **pancreas** is fully formed at birth; during the first three weeks the pancreatic juice has probably no converting action on starch (Korowin). After the twenty-fourth day, Zweifel found a ferment in the pancreatic juice capable of converting starch. This action is supposed to be independent of any action of the intestinal bacteria. The enzyme invertin is found in the wall of the small intestine, and its inverting action on milk-sugar is apparent in the newborn infant (Pautz and Vogel). Lactase capable of inverting lactose is found in the feces of the nursing infant (Orban). Milk-sugar is from earliest infancy fully converted in the gut and absorbed (Czerny).

**The Bacteria.**—Although the morphological and biological characteristics of the bacteria of the mouth, stomach, and intestine have been extensively studied, little is known of their physiological effects on the food during its passage through the gastro-enteric tract (Czerny).

### CHARACTERISTICS OF THE STOOLS OF INFANTS.

This subject has been treated in a general way in the chapter on Hygiene. In addition, it may be noted that the movements of bottle-fed differ from those of breast-fed infants in that they are of a lighter color and in the main more bulky. In a perfectly normal breast-fed infant the movements may at times vary in color and general consistency. Gregor has accounted for this by assuming that the stool of the infant at the breast will vary because the composition of the nurse's milk varies not only from day to day, but also at different hours of the day. The movements of the breast-fed infant will thus at times contain small white or greenish curds or watery elements without the existence of any disturbance of the general health. The character of the movements of bottle-fed infants will vary with the food taken. Infants taking a malted food will have movements that are dry and broken up into crumbs, and have a distinct odor of malt. The feces of the breast-fed infant have a distinctly acid odor, while those of the bottle-fed infant have an odor of decomposition (Czerny). In general the feces of infants may be said to contain digested absorbable substances, indigestible substances, products of digestion and decomposition, anatomical elements of the digestive organs of the stomach and gut, cellular elements, and bacteria.

If the movements of the breast-fed infant are closely examined, they are found to contain small whitish-gray particles, the so-called milk granules of Uffelmann. These were at first thought to be



composed of casein; it is now known that they are made up of fat and fat-crystals or zoöglæa of bacteria. In addition, there are found in the feces of infants epithelial elements, bilirubin crystals, and cholesterin plates. Fat appears in the feces of infants rarely as fat-globules, but generally as fatty acids, neutral fats and soaps. The movements of infants on a mixed diet contain free starch-granules, cellulose, and also cholesterin-plates and bilirubin.

The products of decomposition—indol, skatol, and phenol—are also found according to the time which has elapsed since the voidance of the movement (Blauberg).

Sugar is not found in the feces of infants, or only in small quantities (Uffelman, Blauberg).

Michel has found that the gross weight of feces in the newborn breast-fed infant was about 1.5 per cent. of the gross quantity of food ingested, while later in infancy the movements were 2.7 per cent. of the amount of food ingested. Rubner and Heubner found that in bottle-fed infants the feces were about 7.4 per cent. of the total amount of food ingested.

Michel found that the amount of water in the feces in the first days of infant life was about 72 per cent., while at the ninth month it was 85 per cent. (Uffelman).

### ACUTE GASTRIC DYSPEPSIA.

(*Indigestion.*)

Acute gastric dyspepsia may clinically be divided into two forms, that affecting infants, and that affecting older children. The period of infancy is one of frequent disturbances. Mental excitement on the part of the nurse may cause the milk to disagree with a breast-fed infant. The ingestion of an undue quantity of breast-milk, even if of good quality, may cause indigestion. Certain articles of food, notably asparagus, if partaken of by the mother, may cause gastric irritation. Nursing a breast in which the milk has caked will also cause indigestion.

**Symptoms.**—Vomiting is the first evidence of disturbance of the digestive processes in the infant. It occurs after feeding, and is at first not accompanied by constitutional symptoms or diarrhea. If the exciting cause continues, a slight febrile movement is noted, and also slight prostration. The infant is restless, but having vomited is relieved, and if permitted will again take the breast, the vomiting taking place after each nursing. The bowel movements then become disturbed. They may not only be green, but also frequent and in some cases fluid. There are in all cases colic and tympanites.

Acute gastric dyspepsia in older children may be caused by some article of diet which has disagreed with the patient. The symptoms

are much the same as those seen later in life. It is important both with infants and children to determine whether the symptoms are due to improper food or whether proper food has for some reason disagreed. Bottle-fed infants are liable to indigestion if the milk contains any extraneous substances, not necessarily toxic ones.

A baby may have thrived for weeks on a certain food-mixture, when suddenly, without apparent cause, symptoms of gastric dyspepsia supervene. In such cases it will be found that the acidity of the milk was greater than usual, or that the fodder of the cow has been changed.

**Course.**—If the giving of food is suspended and proper treatment instituted, the symptoms subside and the infant recovers, but if the exciting cause is not removed, more serious disturbance of the stomach and gut will develop.

**Treatment.**—It is best both with breast-fed and bottle-fed infants to discontinue the giving of all food as soon as symptoms of indigestion appear. With the suspension of food the administration of a simple cathartic (castor oil) is all that is necessary. The infant is put for twelve hours on a solution of white of egg, and the breast pumped regularly every three hours to prevent caking. The breast may then cautiously be exhibited. Stomach washing should not be resorted to, and the breast should not be denied for too long a period. If, on resuming breast-feeding, symptoms reappear, an analysis of the milk should be made. Its composition may have changed and too much fat may be present. We should not be hasty in taking an infant from the breast and placing it on the bottle on account of a few symptoms of gastric dyspepsia. Proper regulation of the diet and the taking of proper exercise by the nurse will frequently cause the desired adjustment of the constituents of the milk and the disappearance of symptoms.

### HABITUAL VOMITING OF INFANTS.

By habitual vomiting of infants is meant the regurgitation of milk in the uncoagulated state shortly after nursing. It occurs in infants in apparently good health, and is not followed by loss of weight or disturbance in the functions of the gut. Some infants vomit curdled milk in the same manner. The cause of this form of vomiting has been variously explained. The simplest explanation is, that by slight pressure the food is forced into the œsophagus and thence reaches the mouth. It is a well-known fact that the stomach of the infant can be emptied by gentle abdominal pressure. Another explanation is that on deep inspiration the negative pressure caused by descent of the diaphragm forces a certain amount of fluid from the stomach, which is almost vertical in the infant, into the œsophagus



and thence into the mouth. This form of vomiting requires no treatment. The general impression is that it can be stopped by regulating the amount of breast-feeding, but this belief is erroneous, as the vomiting persists after such precautions have been adopted. Fleischman thinks that the habit is hereditary in certain families.

There are several other forms of vomiting which are of interest in this connection :

*a.* Some children vomit when irritated or after outbursts of temper, or may vomit at will if their food or anything in connection with their discipline does not meet their approval. Some of the little patients know intuitively that vomiting alarms the mother, consequently it will appear whenever any concession is to be obtained in the nursery.

*b.* Vomiting, especially after eating, may be caused by a severe attack of coughing. If vomiting occurs frequently under these conditions, whooping-cough should be suspected.

*c.* The vomiting of pyloric stenosis of the congenital type is characteristic. It is more in the nature of a regurgitation. When lying on the back the baby vomits at intervals, and in small quantities. After a nursing there is an interval, after which the infant vomits two or three times the amount of food taken at the recent nursing. This is explained by the fact that in this condition there is some little vomiting constantly going on, due to the increased peristalsis of the stomach. There is, however, a small quantity of food retained in the stomach. This residual quantity increases with each feeding, and is finally rejected in the manner just described.

*d.* The vomiting of appendicitis is also characteristic. The patient is seized suddenly with sharp abdominal pain and then begins to vomit. The vomiting may recur once or twice, and then cease. In neglected cases, in the final agonal stage, vomiting due to sepsis and toxæmia may be persistent.

*e.* Vomiting is the first symptom in intestinal obstruction. It may be followed by a very small movement, and then for a short time there is, as a rule, no action on the part of the bowels. The vomiting may not recur in the first twenty-four or forty-eight hours, except at long intervals, but the bloody movements recur frequently, and pain is also present. The vomiting returns when the intussusception is more marked, and late in the affection becomes fecal.

*f.* Vomiting occurs at the outset of the infectious diseases. Persistent vomiting extending over a period of months is often of nephritic origin. In connection with the subject of constipation, a form of vomiting of intestinal toxic origin will be described.

*g.* The vomiting which accompanies meningitis occurs at the outset in that of the cerebrospinal type, and is quickly followed by cerebral symptoms. In tuberculous meningitis it occurs at the onset

and after the appearance of a vague series of cerebral symptoms. It is rarely persistent after the initial attack. The subsidence of the vomiting and the sequence of cerebral symptoms and a febrile movement will easily distinguish this form of vomiting from others.

Tumor and abscess of the brain are accompanied by vomiting at intervals.

### COLIC.

Colic is not a disease, but a symptom of disturbed conditions of the gut. It is really a painful contraction of the muscle-fibre of portions of the gut-wall. In the simplest form the painful contractions are incited by actual distention of the lumen of the gut. The pain caused in colic is in the majority of cases not of the character which arises in certain other affections of the gut which are neurotic in nature, nor is it of the same nature as that seen in enteritis. Pain similar to that in colic may be caused by the administration of some such drug as lead, arsenic, etc.

**Cause.**—In the great majority of cases the affection is caused by some disturbance of the processes of assimilation in the gut. It is uncommon in infants in good condition, and its appearance in any case indicates the necessity of an investigation into the condition of the digestive processes in the stomach and intestine. The form of pain or colic accompanied by distention (tympanites) seen in newborn infants, and also at the height of pneumonia in older children, has an etiology distinct from that of the ordinary variety. Not only is the pain of neurotic origin, but also the distention is a result of paralysis of the muscular fibre of the gut. The processes in the gut may be disturbed as a result of the pneumonia. Colic may occur in breast-fed or in artificially fed infants. In the former it is not always possible to discover the exact cause. The breast milk may be abundant, of good color, and of correct composition, and still there may be very violent colicky pains. In artificially fed infants the cause of the colic may lie in the very nature of the food (cows' milk) and the difficulty of complete assimilation. Thus not only will an excess of proteids in the milk cause colic; the nature of the proteids of cows' milk, no matter how much they are diluted, will cause colic. An attack of colic is preceded by general uneasiness; the infant cries and cannot be quieted. The severe colicky pain is accompanied by sharp cries, the arms and lower extremities are drawn up, and the abdomen is rigid. After the passing of gas the infant is quieted and falls asleep quite exhausted. These attacks of colic deprive the infant of sleep; they may or may not be accompanied by tympanites. The movements are merely normal, or may be normal for some days and then take on





PLATE XVI.



So-called Idiopathic Abdominal Distention or Tympanites in an infant  
nine days old; recovery.



a curdy character or become greenish. Sometimes the colicky attacks are accompanied by a mild form of diarrhoea; the pain may be so severe as to cause convulsions.

### TYMPANITES.

Tympanites is a condition of distention of the gut with gas, which may supervene in inflammatory states of the peritoneum. In such conditions (peritonitis, appendicitis) the paralysis of the muscular wall of the gut is the real cause of the distention. In other states, such as pneumonia, it may be the result of inefficient action of the diaphragm and of an enteric catarrh which sometimes accompanies that disease. In the newborn infant, tympanites is a result of an inherent muscular weakness of the intestinal wall (Plate XVI.). In colic due to imperfect assimilative processes in the gut, the tympanites is due to the formation of gases of which the intestine is unable to rid itself rapidly.

In pneumonia the tympanitic distention is sometimes extreme, causes great distress, and is frequently mistaken for peritonitis. In the forms of distention in the newborn infant the distress is not so great. In rachitis there is a state of tympanitic distention of the abdomen due not only to defective assimilative processes, but also to a lax condition of the muscle-fibre of the intestinal walls.

**Treatment of Colic and Tympanites.**—If the food of a bottle-fed infant is at fault, the modification of milk must be altered so that the proportion of the proteids may be lower. A reduction of proteid will not always remedy the condition; the proportion of sugar is sometimes at fault, especially in infants fed on condensed milk. Not more than 6 per cent. of sugar should be added to any milk modification. Some infants can take a large quantity of malt-sugar in their food and not suffer from colic. If a breast-fed infant suffers from colic, the hygiene of the nurse should be attended to. If after the taking of exercise and regulation of diet the colic persists and becomes a feature in the case, the wet-nurse should be changed.

The attack of colic is best combated by giving the infant an enema. In some cases a small amount of dilute hydrochloric acid and pepsin given three times daily will alleviate the symptoms. If in spite of all efforts an artificially fed baby suffers with colic and does not increase regularly in weight, it should be placed at the breast.

### HYPERTROPHIC PYLORIC STENOSIS.

*(Congenital Stenosis of the Pylorus; Congenital Hypertrophy of the Pylorus and Stomach-wall; Congenital Gastric Spasm.)*

Hypertrophic pyloric stenosis is a congenital condition appearing from a few days to several weeks (three months) after birth, and

manifesting itself in persistent vomiting. In a few instances several infants in the same family have been thus affected.

The **etiology** of the affection is obscure. Since in the majority of the cases which have been carefully studied the infants were overfed or improperly fed, it is supposed that some irritant to the stomach is the exciting cause. Thomson, who has made careful studies of these cases, believes that the condition originates in intra-uterine life, and is due to the ingestion of liquor amnii. This fluid, by irritating the mucous membrane of the stomach, excites both that organ and the pylorus to overaction. Pfaundler, voicing the teachings of Escherich's school, denies that there is a true hypertrophy of the pylorus, and asserts that the condition during life is that of functional spasm. The post-mortem condition is due to toxic agonal contracture of the pylorus.

**Morbid Anatomy.**—The stomach and œsophagus have been found to be dilated in fully one-third of the reported cases. The mucous membrane shows the usual changes, such as the congestion which is seen in a stomach in which there have been functional disturbances. The mucous membrane of the pylorus is thrown into voluminous folds. The lumen has in some cases been found patent to a small probe, but fluids cannot be forced from the stomach into the pylorus (Thomson). The muscular fibres show characteristic change. The circular fibres are thickened and hypertrophied (Thomson). In Finkelstein's case the longitudinal fibres were also thus affected.

**Symptoms.**—Infants in whom this condition is present are of normal weight and appearance when born. As a rule, the desire for food is greater than is normal. In the majority of cases the infants are allowed, on account of supposed insufficiency of the breast milk, to nurse an excessive length of time. It is soon noticed that there is vomiting of small quantities of milk after each nursing. After a few days all the food taken into the stomach is rejected, and the vomiting attacks increase in frequency. The vomited matter never contains bile-stained matter. At times it is less than or about equal in amount to the quantities ingested at the nursing. At intervals attacks of vomiting occur during which more food is rejected than has been taken at the preceding nursing. This proves that there is not only retention of food, but also lack of absorption by the mucous membrane of the stomach.

The infants rapidly lose weight; the abdomen has a characteristic appearance in all the cases I have met. The abdominal walls are lax, the coils of gut can be clearly made out, and the peristaltic movements are visible. In the epigastrium, just beneath the ribs, is a large coil, and to the right of this a constricting band and what appears to be another coil. These are in constant peristalsis. I have seen these coils in two cases, and in both they had



been mistaken for the stomach. They were, in my opinion, coils of the transverse colon. In two of my cases I could on deep palpation in the median line, midway between the ensiform cartilage and the umbilicus, feel a resistant nodular mass, which may have been the contracted pylorus (Thomson and Finkelstein). I have not been able to map out a dilated stomach.

**Prognosis.**—If the vomiting is unrelieved, death occurs in from three weeks to six months. On the other hand, all of these cases are not hopeless. Heubner has seen three cases (Finkelstein) which recovered. I have seen three cases in which the history, symptoms, and physical signs were undoubtedly those of congenital stenosis, and all of which recovered. One case gained to a remarkable degree in weight, another in three months contracted gastro-enteritis and subsequently a terminal intussusception and died unrelieved; the third recovered.

**Treatment.**—All mouth-feeding should be suspended. The infant is kept in the recumbent position, and the stomach washed out once; all binders and constricting clothing are removed from the abdomen. The infant is then fed by the rectum for one or more days with small quantities of albumin-water, or an ounce of somatose solution is introduced per rectum three or four times daily. This amount, although seemingly small, will if retained, sufficiently nourish the patient. The stomach is thus given complete rest. After two days teaspoonful doses of albumin-water are given by mouth every hour, and are supplemented by rectal enemata. If the vomiting has diminished or ceased, artificially fed infants are given by mouth a very dilute milk modification (0.5 of proteids, 2 of fat) in half-ounce doses, pasteurized or sterilized. The milk is given alternately with albumin-water. Milk is at first given only three times in the twenty-four hours alternately with albumin-water. If vomiting recurs, mouth-feeding is again suspended and rectal alimentation substituted. The infant is not moved, especially after being fed. In this tentative way, gradually increasing the quantity and strength of the milk (1.2 of proteids, 2.5 of fat), I have succeeded in three cases in effecting tolerance of food and cessation of vomiting.

Medicines are of little value in these cases. I have used small doses of bismuth subnitrate in combination with pepsin, to allay the irritability of the stomach.

**Surgical Treatment.**—Two surgical procedures have been proposed and attempted in these cases. The first method is that of opening the abdomen and establishing a communication between the stomach and gut by means of a Murphy button. This procedure, first suggested by Schwyzer, was carried out by Willy Meyer on the case of Meltzer, with fatal results. The second method is that adopted by Nicoll in the case of Ritchie. The abdomen was opened, an incision made near the pyloric end of the stomach, the pylorus

forcibly dilated with forceps, and the wound closed. The infant recovered. This latter is the only recorded case of recovery after operation. In view of this fact, I believe that with these weak infants every expedient should be tried before resorting to surgical intervention.

## ACUTE GASTRO-ENTERITIS

(including *Cholera Infantum*).

(*Summer Diarrhœa; Acute Gastro-enteric Infection.*)

Acute gastro-enteritis is a form of diarrhœa usually accompanied by gastric symptoms. It is prevalent in the summer, but may also occur during the winter months. Bottle-fed infants are more subject to the affection, although it occasionally attacks infants at the breast. In institutions epidemics of gastro-enteritis occur in breast-fed infants. In large cities more than one-half the deaths among infants under the age of twelve months are caused by summer diarrhœa. In Paris, Chaterinkoff found that of 20,000 children dying of gastro-intestinal disorders, fully three-fifths were bottle-fed. This high rate of the mortality of bottle-fed infants, as compared with that of breast-fed infants, is not alone due to the difference in the nature of the food; no matter how carefully it is handled before it reaches the infant, milk passes through many channels, and in each of these it is exposed to infection. The intense heat of summer also favors the increase of infectious agents.

**Classification.**—The various forms of acute gastro-intestinal infection may be divided into those whose source of infection lies outside the body (ectogenous) and those in which the elements of infection are pre-existent in the body (endogenous). This classification (Escherich) of the diarrhœa of infancy is both practical and in accordance with the results of recent study. In the first class are included the diarrhœas of toxic origin and cholera infantum; in the second are included the diarrhœas which are caused by varieties of bacteria pre-existent in the gut, but which, in the opinion of Booker, Escherich, and Marfan, may under favorable conditions increase to enormous numbers and become virulent. According to Booker, no one specific micro-organism is the essential cause of gastro-enteritis or acute summer diarrhœa. Escherich has shown that the coli group may under certain conditions become virulent. Of the bacteria which are found in certain forms of gastro-enteritis, the *Streptococcus enteritidis* seems to have attracted the greatest attention. Booker first insisted on the importance and peculiar rôle of this micro-organism. He found these streptococci in great numbers not only in the stools of infants suffering from acute summer diarrhœa, but also in the walls of the gut and in the various organs



of the body. Escherich, Libman, and Hirsch have confirmed the results of Booker. Escherich regards the *Streptococcus enteritidis* as an ectogenous infection. The udder of the cow may be the source of this micro-organism. Marfan and Booker are also inclined to believe that streptococci are able under certain conditions to increase in number and virulence and that they are one of the endogenous forms of infection by a micro-organism normally present in the gut. Among the other bacteria found in enormous numbers in the movements of infants and children suffering from acute gastro-enteritis are the *Bacillus pyocyaneus* (Kossel and Baginsky), *Proteus vulgaris* (found by Booker in choleraform diarrhœa), and the proteolytic bacteria, or the ferment of the casein of the milk. The last class comprises peptonizing bacteria, such as the *Bacillus subtilis*, *Bacillus mesentericus vulgatus*, and *Tyrotrix tenuis*. These peptonizing bacteria are not found in the gut or stools of the breast-fed infant either when in good health or sick. We may thus classify all diarrhœas of acute gastro-enteritis as follows:

1. Those due to improper food, or the so-called mechanical irritative diarrhœas (Booker).

2. The infectious form of gastro-enteritis (endogenous and ectogenous). This class would include the toxic diarrhœas of some authors.

Not only the food and the bacteria, but also certain changes in the gut play an important rôle in acute gastro-enteritis.

**Morbid Anatomy—Stomach and Intestines.**—Booker has described a superficial loss of the epithelium of the stomach and gut, as a constant lesion in all fatal cases of gastro-enteritis. It may be intact in some places and destroyed or eroded in others. The mucous membrane of the jejunum and duodenum may show less denudation than other parts of the gut. The epithelial layer of the mucosa is infiltrated with leucocytes in diffuse areas or nests. The infiltration may push the epithelial layer upward. The mucosa itself is infiltrated with polynuclear and mononuclear leucocytes to a varying extent. The mucosa shows superficial or deep ulcerations involving the crypts or villi. Heubner has described a form of necrosis which chiefly affects the epithelial structure without involving the deep mucosa. This occurs in cholera infantum. Booker also describes a bronchitis and a form of bronchopneumonia which are quite constantly found in fatal cases of gastro-enteritis. Hemorrhages into the lung tissue are common.

In the **kidneys** there is necrosis of epithelium in the convoluted and irregular tubules (Booker).

The **liver** shows fatty degeneration and necrosis of the liver-cells. The **lymph-nodes** show focal necrosis.

**The Role of the Bacteria.**—Booker has demonstrated that no bacteria are found in the mucosa of the intestine if the superficial

epithelium is intact. If there is a lesion of continuity of the superficial layer, the bacteria invade the mucosa in large numbers. There is reason to believe that the toxins generated by the bacteria in the gut cause the superficial erosions and prepare the way for invasion of the lymph-channels and bloodvessels. Bacteria are not always found in the lesions, but as a rule the ulcerations of the mucosa show vast numbers. Booker found bacteria in cultures taken from the solid organs and blood, thus confirming what Czerny and Mozer found to be the case during life. The lungs especially showed large numbers of bacilli and cocci.

**Symptoms**—In the mild form of gastro-enteritis the infant is restless and cries at intervals because of colicky pains. It may previously have been in good health, but with the advance of these symptoms there will also be noticed a slight febrile movement and a disinclination to take the bottle or breast. Vomiting occurs after feeding, the rejected contents of the stomach being curdled and having a marked acid odor. In mild cases the vomiting is usually not severe. It may be repeated three or four times in the twenty-four hours. The movements are at first normal; they afterward become frequent and contain whitish curds or greenish and white curds, are more fluid than is normal, and may have a very offensive odor. In mild cases there may be only two or three such movements in the twenty-four hours or they may number six or more. Later, the fever also becomes more marked, the temperature sometimes mounting as high as 103° F. (39.4° C.). If the feeding is continued, the vomiting persists. The infant shows little or no prostration.

In severe cases the vomiting is marked from the outset. The infant not only vomits its regular food, but will also often vomit all fluid that is taken into the stomach. The diarrhoea is also more severe than in the mild forms. The movements are at first yellow or greenish and contain white curds, but as the disease advances they become more fluid, until in very severe cases only a greenish malodorous liquid containing small particles of mucus and fecal matter is voided. The infant has a febrile movement which varies from 101° to 103° F. (38.8° to 39.4° C.), and there is marked prostration. In the acute forms of gastro-enteritis there is considerable loss of weight; the infant becomes pale and languid, and the pulse is rapid and weak; the number of daily evacuations may reach twenty. In some cases the straining causes a descent of the lower part of the rectum, and the movements contain a slight amount of bloody mucus. The odor of the evacuation may not be offensive. If the patient improves, the symptoms retrograde—the vomiting becomes less frequent, the stools more fecal in character and less numerous, and the fever subsides. If, on the other hand, the symptoms progress, the movements not only continue frequent and fluid, but also blood and particles of mucus are mingled



with the fecal matter. The vomiting may cease entirely. The infant loses in weight steadily; the movements are small and passed with tenesmus; the patient passes into the subacute stage of gastro-enteritis. In some cases there is colic; the infants are restless or pass into an apathetic condition. Little urine is passed, and in the majority of cases of mild or severe gastro-enteritis, albumin is present. It rarely amounts to more than a trace. In severe cases there are leucocytes and epithelial, hyaline, and blood-casts in the urine; sometimes in addition a few blood-cells are found.

In the subacute forms of gastro-enteritis which last for more than a week, bronchopneumonia may be a complication. This form of bronchopneumonia is described in the section on Pneumonia. In some cases it is of short duration, in others persistent. Bronchopneumonia with slowly resolving areas of consolidation in the lung is the type met with.

**Course and Prognosis.**—The prognosis of the mild forms of gastro-enteritis is good, if proper measures are adopted. The severe forms are exceedingly fatal in summer. The mortality varies with the environment. In the crowded tenements of large cities and in unhygienic surroundings the mortality is great, as is also the case in institutions and hospitals. In private practice the isolation of the patient and special nursing reduce the mortality to a minimum by preventing reinfection. Reinfection is caused by lack of care in handling the diapers and in preparing the food, by giving improper food, and by placing a number of cases in the same room. There can be no question that in hospitals patients are affected unfavorably by proximity to other patients suffering with the same disease. No matter how careful the nursing under such circumstances, reinfection cannot be prevented. Also, perfect cleanliness is not so nearly attainable in hospitals as in private practice.

### **Cholera Infantum.**

Cholera infantum is the severest form of summer diarrhœa prevalent among infants. It is believed that it has a specific origin, but this has not as yet been demonstrated. Cholera infantum does not occur so frequently as has been hitherto supposed. Of hundreds of cases of gastro-enteritis of the acute variety which come under my care yearly, only a few can be called typical of this form of infectious diarrhœa. These cases occur for the most part in weakly bottle-fed infants. Breast-fed infants may occasionally be affected, especially in hospitals.

**Symptoms.**—The infants as a rule have been suffering from a mild diarrhœa. Following a slight febrile movement, vomiting and diarrhœa of a severe and exhausting character set in. The bowel movements are frequent, but contain very little fecal matter after the

first few have been passed. They are at first greenish, afterward becoming watery, resembling barley-water; they contain but a few flocculi of mucus, and may not have much odor. The vomiting is incessant. First the stomach contents are vomited, and finally a greenish fluid. Within a few hours the infant is reduced to a condition of great prostration. The loss of weight is marked, even in the first twenty-four hours. The skin on the thighs is wrinkled.

The face and trunk are pale and the face is drawn. There is fever to a marked degree ( $101^{\circ}$ – $103^{\circ}$  F.,  $38.3^{\circ}$ – $39.4^{\circ}$  C.), and the pulse is rapid and thready. Toward the close the movements are passed involuntarily. The whole picture is that of a choleric form disease. As the fatal issue approaches the eyes become sunken and glassy, the fontanelle is depressed, and the mouth is open. The condition described elsewhere as hydrencephaloid sets in. Convulsions and a rise of temperature ( $105^{\circ}$  to  $107^{\circ}$  F.,  $40.5^{\circ}$  to  $41.6^{\circ}$  C.) precede the fatal issue.

**Occurrence.**—These severe choleric form diarrhoeas resemble Asiatic cholera very closely, and should be sharply differentiated from severe forms of gastro-enteritis. They occur in bottle-fed infants under the age of two years, and chiefly in the months of July and August. Heat and infected food are the main etiological factors. A diarrhoea of a mild type is the forerunner in the majority of cases. These cases are not so frequent to-day as they were in the days when infants were fed with decomposed milk containing bacterial toxins. This form of diarrhoea must therefore be looked upon as a purely ectogenous infection.

**Duration and Prognosis.**—The prognosis in the majority of cases of cholera infantum is grave. The disease is an exceedingly fatal one, occurring as it does for the most part in infants fed on the bottle and whose general condition is poor. It lasts for from twenty-four hours to two or three days. The rapidity of the development of the symptoms and of the fatal results precludes the possibility of any complications other than those due to the great drain on the system. The condition of hydrencephaloid is hardly a complication; it is a terminal set of cerebral symptoms. Sclerema, mentioned by some authors, I have not met in true cholera infantum; it is seen in the terminal stage of acute forms of gastro-enteritis. This form of sclerema affects the thighs at the upper and inner part. It is described in the section devoted to that subject.

Kjelberg, Felsenthal, Bernard, Morse, and the author, found albumin and casts in the urine of children suffering from all forms of gastro-enteritis, acute and subacute, including cholera infantum.

Morse as well as the author found that the urine was concentrated and contained hyaline, granular, and epithelial casts, with leucocytes and blood-casts. The albumin is rarely present to a marked degree. It is a trace or a distinct reaction. The urine is



suppressed in severe cases, and lessened in quantity in others. In some cases of gastro-enteritis of the severe types there is slight œdema of the subcutaneous tissues, especially on the inner part of the thighs, the legs, and dorsum of the foot. We are not in a position to trace any close relationship between the general symptoms and the disturbances of the kidney. The toxæmia in this disease, causing as it does vomiting and nervous symptoms, masks the nephritic symptoms if they are present.

The **diagnosis** of acute gastro-enteritis is not difficult. There are, however, many infectious diseases, the onset of which closely resembles that of an attack of gastro-enteritis. Scarlet fever, for example, begins with vomiting, and in some cases with diarrhœa. There is a form of grippe which in its onset, with vomiting and diarrhœa, closely resembles an attack of gastro-enteric disease. In fact, these symptoms may persist in the course of the former affection.

The physician should not be satisfied with a history of gastro-enteric symptoms, but should carefully examine the skin, throat, and chest at every visit. In the severer forms of diarrhœa a small particle of the movement may be spread on a cover-glass and examined for an excessive number of streptococci. In mild forms of diarrhœa we should not fail to make a Widal test of the blood and a count of the leucocytes, to eliminate the possibility of typhoid fever. This will especially be indicated in cases in which there is enlargement of the spleen.

**Treatment of Acute Gastro-enteritis and Cholera Infantum.—**

**Prophylaxis.**—The nursing bottles when emptied by the infant should be filled with a saturated solution of sodium bicarbonate, allowed to stand for a few hours, and then carefully washed inside and out with a bristle brush. The nipples should be sterilized daily. The nurse or mother, after attending to the diapers of the infant, should carefully cleanse the hands before feeding the baby. The milk should be diluted as directed in the section on Infant Feeding, pasteurized or sterilized, and then kept on ice until needed. It should be obtained from a dairy in which cleanliness of the utensils and in the milking of the cows is observed in all details. The milk should be fresh and delivered for modification within a few hours of the milking-time. The nursings should be conducted at stated intervals. If there is a residue in the nursing bottle, it should not be utilized for a subsequent nursing. The infant is given a full bath daily. By attending to all these details, infection of the food and of the infant may be avoided. With breast-fed infants prophylaxis is of great importance. A baby at the breast should be fed at regular intervals. The breast-nipples should be washed with a saturated solution of boric acid before and after nursing. The baby should not be allowed to nurse a breast with a fissured nipple.

The milk of such a breast is pumped off, and an attempt is made to heal the nipple in the manner elsewhere described. If there is caking of the breast, the condition should be remedied before the infant is allowed to nurse. Abundance of fresh air and bathing are indicated in these infants as in bottle-fed infants.

**Sick Infants.**—As soon as a baby shows signs of even mild dyspepsia or gastro-enteritis the milk should be discontinued, a simple cathartic given, and the infant kept for twenty-four hours on a solution of egg-albumin. Vomiting which has occurred only once or twice does not call for active treatment, as it will disappear as soon as the milk is discontinued. After the bowels have moved, if the infant shows no exacerbation of symptoms feeding should be resumed cautiously. In this way a severe gastro-enteritis can be averted. If the food is not suitable, causing signs of dyspepsia such as colic, it should be changed if possible, else severer symptoms may result. If in spite of all precautions an attack of gastro-enteritis develops, the patient should be treated on the following lines :

1. The food is stopped and another of a safe character substituted.
2. The toxins are eliminated and the strength of the patient supported by the so-called mechanical methods.
3. Drugs are used to abate the symptoms and support the strength of the patient.

The milk, whether of the breast or bottle, is discontinued. The infant is given a solution of albumin-water, acorn-cocoa, or beef-juice expressed and diluted with barley-water. A baby can be kept for days upon these mixtures without any danger of reducing the strength. The white of one egg is equal in nutritive value to three ounces of breast-milk. It is digestible, and is well borne by infants. Albumin-water may be used alternately with the solution of acorn-cocoa or beef-juice and barley-water. To older children who are suffering from gastro-enteritis we may sometimes have difficulty in administering albumin-water or acorn-cocoa. Under such conditions I frequently resort to a dextrinized gruel or the so-called Liebig's soup mixture which Keller advised. I find that after the acute symptoms are past, infants and children who refuse every other form of food will take this with eagerness. It may be given while the diarrhoea is still in progress, but should not be given until the vomiting has ceased.

The cathartic given at the onset should be castor oil or calomel, grain ss (0.03) doses twice or three times a day. Infants who are vomiting are given calomel in preference to castor oil.

**The Vomiting.**—If the vomiting is not severe and the case is under treatment from the onset, it is best not to wash out the stomach at once. It often happens that the vomiting ceases as soon



as the regular food is stopped. If, however, the vomiting persists for twenty-four hours, we proceed to wash out the stomach once. If the vomiting continues after this, it is either toxic or may in rare cases be due to some other causes. As a rule, it ceases after one irrigation of the stomach.

The *diarrhœa* is controlled by irrigation of the gut. The rectum and gut are washed out in those cases in which the *diarrhœa* is not only persistent, but progressive. The object in washing out the lower bowel in any form of acute gastro-enteritis is twofold: (a) to remove any residue of feces that may have collected in the lower bowel and rectum, and to stimulate peristalsis and thereby favor evacuation from above; (b) to stimulate the heart and add to the body an amount of normal solution to compensate for the drain caused by the *diarrhœa*. The Cantani normal salt solution is utilized in the manner described on page 42.

The rectal enemata are given under a pressure obtained by an elevation of at most two feet from the bed. A temperature of  $107^{\circ}$  to  $110^{\circ}$  F. ( $40.5^{\circ}$  to  $43.3^{\circ}$  C.) is the best and most stimulating in these cases. Fully a quart of water is thrown into the rectum in half-pint portions. As the half-pint flows in, the funnel on the rectal tube is disconnected and the contents of the bowel are allowed to escape. Another portion is then allowed to flow into the bowel. The water will sometimes escape alongside of the tube. This is rather a favorable sign, being significant of the contractile powers of the gut and abdominal walls. Only two enemata daily are necessary, even in severe cases of acute gastro-enteritis. As the *diarrhœa* and symptoms subside we reduce the number of enemata to one, finally discontinuing them entirely as the infant improves. It sometimes happens that after a few days the enemata are followed by movements containing blood and mucus, the tenesmus being aggravated. In these exceptional cases an enema must be given only every other day, and the effect on the rectal discharges watched. By stopping the enemata altogether it can be determined whether the discharges of mucus and blood are caused by the therapy or the disease.

**Hypodermoclysis.**—The injection of normal salt solution under the skin is indicated only in the severe cases of acute gastro-enteritis in which, as in *cholera infantum*, the course of the disease is rapid and the prostration extreme. Personal experience rather discourages the employment of large injections by this method. I have seen two cases of infection by the *Bacillus capsulatus aërogenes* (Welch) following hypodermoclysis. These occurred through the use of saline solution evidently insufficiently sterilized, and which had probably been allowed to stand before being used. In a third case hemorrhages over large areas occurred at the point of the injection of the salt solution. These injections are also very

painful. Because of these dangers and disadvantages the subcutaneous injections of salt solutions should be utilized as a last resource in desperate cases. Small rather than large amounts of fluid should be injected subcutaneously, as was advised on page 36. The salt solution for the hypodermoclysis is that of Cantani. It should be sterilized at a temperature of 212° F. (100° C.) for at least an hour, to kill sporulated bacteria if possible. A very fine hypodermic needle is used in the manner described on page 36.

**Baths.**—In all cases of acute gastro-enteritis, whether with or without elevation of temperature, the benefit obtained from warm baths cannot be overestimated. In cases of great prostration a bath of 108° F. (42.2° C.) for five minutes is stimulating to the nervous centres and is followed in many cases by diminution of the apathy and an apparent reduction of the effects of toxæmia. If the temperature rises above 103° F. (39.4° C.), sponging with water at 80°–85° F. (26.6°–29.4° C.) is all that is needed. This should not be done oftener than once in every three hours.

**Alcohol.**—Of late years, whiskey is given less and less in cases of acute gastro-enteritis. In these cases there is a special intolerance of the stomach and also of the economy to whiskey. Infants after taking it for twenty-four hours will become stupid, apathetic, and exhibit a constant retching if they do not vomit. This appears to be due more to the effect of the alcohol locally on the stomach and also systemically than to toxæmia of the disease. I therefore deprecate the use of alcohol except in extreme cases, when whiskey is given in small doses at short intervals.

**Strychnine** is useful for its stimulating effect on the heart. Grain  $\frac{1}{800}$  (0.0002) is given to an infant of six months, and grain  $\frac{1}{200}$  (0.0003) to older infants every three hours.

**Atropine**, lately advised as a cardiac stimulant in these cases, especially in cholera infantum, is a dangerous drug, and should not be employed. I have seen grain  $\frac{1}{150}$  (0.0004) give rise to constant tremulous and convulsive twitching.

**Resorcin.**—If the vomiting is constant, grain  $\frac{1}{8}$  (0.008) of resorcin given every three hours is a safe and very useful remedy. It should never be used in larger doses nor at shorter intervals.

**Bismuth** in the form of the subcarbonate is the only drug useful in allaying the vomiting and the tenesmus of the bowel. Grains ij or iij (0.12 or 0.18) are given in powder form every two or three hours.

**Opium** in any form has fallen into disuse. In the severe cases it is dangerous, and may increase the prostration; in the milder cases its use is justifiable only if the colicky pains are excessive. The milder preparations such as the wine and the camphorated tincture are of value, because they can be given in graduated doses, and the effects determined more exactly than can be done with the stronger preparations.



**Salol** in grain  $\frac{1}{2}$  (0.03) doses every three hours may be combined with the bismuth to allay the colicky pains.

**Tannigen** is a useful drug in the chronic forms of intestinal disease, but an irritant in the acute forms.

**Colic** has been mentioned so often that a few words as to the treatment may not be out of place. Passing of the rectal tube rarely relieves it. A small rectal enema has been found to be a very effective remedy.

As the symptoms improve care should be taken not to return to a milk diet too quickly. The milk is given in dilutions and is sterilized carefully. Infants in an enfeebled condition as a rule bear this form of milk best, since it is not apt to be irritating to the gut. When the danger is past any form of milk may be given—raw, pasteurized, or sterilized—care being taken that all the precautions as to freshness, cleanliness, and proper preparation are observed. I have mentioned the fact that before returning to dilutions of milk the exhibition of dextrinized gruels has been successful with very weak infants. The malt, the cereal, and the milk acted upon by the ferment contained in these mixtures are all easily digestible and assimilable, and promote increase of weight. As a matter of course, the effect of the gruel mixture on the stomach and gut should be carefully studied.

Whatever methods are employed in the treatment of acute gastro-enteritis, it is necessary to avoid the error of overtreatment. It should be remembered that hours of rest do more than hours of treatment. Three-hour intervals should elapse between the application of remedial measures. Fresh air in the room or a sojourn of a few hours in the open with absolute quiet, is of the greatest value in these cases.

### GASTRO-INTESTINAL ATROPHY.

(*Marasmus*; *Athrepsia* (Parrot); *Simple Infantile Atrophy*; *Chronic Gastro-intestinal Catarrh*.)

Atrophy may follow or complicate congenital syphilis or any subacute or chronic disease of the gut. Gastro-intestinal atrophy, or the athrepsia of Parrot, is a condition due to a faulty operation of the assimilative processes in the gut.

The **etiology** of chronic atrophy is unknown. Keller, who has studied this condition extensively, believes that in infants and children suffering from gastro-enteric catarrh there is an excessive production in the gut of acids which under normal conditions are neutralized. In disturbed conditions, instead of urea, ammonia is excreted in the urine. The formation of the ammonia entails a drain on the economy—hence the emaciation. Excess of albumi-

noids and fats in the food favors overproduction of acids in the gut. In marasmus there is an acid intoxication of the economy, originating in the gut. There is no doubt that under unhygienic conditions and in overcrowded hospitals infection of one patient by another may take place. No satisfactory explanation of the manner in which such infection occurs has as yet been advanced. Heubner has surmised that the excreta of one patient in some way contaminates the food of another by transmission through the nursing personnel. This is true in a certain number of cases, but not in all. Cases of marasmus seen in private practice show progressive emaciation in spite of the fact that the utmost care is exercised in the preparation of food and the strict maintenance of hygienic conditions.

**Morbid Anatomy.**—The body is much emaciated; the skin hangs in folds on the extremities, and presents hemorrhages and petechiæ. The lungs may show atelectatic areas or may be the seat of bronchopneumonia. The heart is small and the muscle-fibre pale. In many cases the stomach is dilated and the mucous membrane pale. The small intestine shows few changes. The Peyer's patches may be slightly raised and show the so-called shaven-beard appearance. The follicles of the colon may be slightly prominent. The microscopical changes in the gut are characteristic. In some places the follicles are the seat of catarrhal inflammation. Both in the stomach and the intestines there are patches where there is an absence of glandular tissue; in its place is a newly formed connective tissue composed of round and spindle-shaped cells. The villi of the gut have disappeared. The whole mucosa is thinner than is normal (Baginsky). On the other hand, these changes may not be marked. The liver is fatty and may be enlarged. The spleen is small. The kidneys may be pale, especially in the cortex, and may be the seat of parenchymatous degeneration. The lymph-nodes of the mesentery may be enlarged.

**Symptoms.**—Cases of gastro-intestinal atrophy are seen among the better classes, but, as a rule, they form a large contingent of dispensary cases exposed to unhygienic surroundings, and often improperly fed. The infant may have been small at birth or prematurely born. In some cases, especially in families in good circumstances, the baby may have been put at the breast and have done well up to the time when, for some reason, it was put on condensed milk or milk which had been carefully modified. In other cases the infant may from the outset have been fed on the bottle with good results up to a certain point, when the weight became stationary and the infant retrograded and showed signs of atrophy. The atrophic course having once begun, the symptoms vary little. There is no gain in weight. The skin, especially about the inner parts of the thighs, becomes wrinkled, and the subcutaneous adipose tissue



diminishes in amount. The extremities lose their normal plumpness. The face has a pinched appearance. The chest is emaciated and the ribs show plainly. The fontanelles, if still open, may be somewhat depressed. Over the buccinator muscles is seen a small cushion of fat, the so-called "sucking pads," which remains when all the other fat has disappeared. This gives to the face a peculiar appearance that is typical of cases of atrophy. If the infant is not well taken care of, erosions and aphthæ are seen on the mucous membrane of the mouth and gums, and there may also be sprue. Even in well-cared-for infants the buttocks may be slightly eroded. If the infant has been neglected, there is marked intertrigo. The buttocks are emaciated and the tuber ischii show prominently. During the progressive emaciation the infants have constant gastro-enteric disturbances. There are colic and at times constipation, or diarrhœal movements alternating with constipation. The movements of the same infant vary greatly. They may be greenish with white curds, are sometimes exceedingly offensive, and at other times may be normal and alternate with slightly diarrhœal movements. The temperature is normal or slightly subnormal. During exacerbations of the intestinal disturbances, it may rise slightly. These disturbances of the gut do not seem to be influenced by changes in the diet. The infants in many cases finally lose all desire for food. Others drink with avidity, but do not assimilate the food taken. If untreated, these infants emaciate until they are reduced to skin and bone. They grow exceedingly weak, and die with some intercurrent infection, such as pneumonia, tuberculosis, or infectious disease.

**Treatment.**—If an infant suffering with chronic atrophy comes under treatment at from the third to the fifth month, the proper procedure is to place it on good breast milk. If this is not feasible and the bottle is the only resort, the task is more difficult. In rare cases carefully modified cows' milk (with the formula of proteids, 1.2; fat, 2.5; sugar, 6) will give the desired results if the infant has not previously had a correct diet, but has been given a proprietary food or a condensed milk formula. In my experience in ambulatory cases, no milk formulæ of any kind have been universally successful. I have found that many of these cases if put on a gruel and milk diet, according to the method described by Keller, and detailed elsewhere in this work, do well: the weight increases, the colic disappears, and the character of the stools improves. After the weight reaches a certain point the milk gruel is discontinued and the infants continue to do well on an ordinary milk formula. In certain cases of marantic infants of the age of twelve months this method has been very successful, especially with those whose distaste for the ordinary milk foods gradually increased. It is necessary to study out the method of feeding which seems likely to be best adapted to the individual case. Greater success can be attained in private prac-

tice than in hospitals. With the feeding, the general hygiene of the infant should receive attention. Daily baths with sea-salt and open-air life are especially indicated.

In infantile atrophy the medical and mechanical treatment are of less importance than the selection of proper food. For this reason we should not seek to multiply remedies. The movements of the bowels in some cases have an exceedingly fetid odor. The treatment is begun with the administration of brisk cathartics, such as castor oil. The bowel is then washed out once a day in the same manner as in gastro-enteritis until the character of the movements has improved. If there is a tendency to diarrhœa, tannigen, with or without bismuth, may be given three or four times daily. If there is any great amount of gas generated in the stomach, a very small dose of dilute hydrochloric acid and pepsin should be given daily after a feeding.

### ACUTE AND SUBACUTE ENTEROCOLITIS.

(*Enteritis Follicularis; Enteric Catarrh.*)

Enterocolitis is peculiarly a diarrhœal disease of infancy and early childhood. It was formerly classified as a form of dysentery, because in these cases the movements are tinged with blood and contain mucus. The cases are, however, really of a milder type, and present many symptoms foreign to true dysentery.

**Etiology.**—In many of its features this affection resembles acute and subacute gastro-enteritis. It is prevalent during the summer months. It occurs in infants after the first year of life, and may be primary or follow an ordinary dyspeptic diarrhœa, an exanthema, pertussis, or bronchopneumonia. Booker has described the great number of streptococci found in certain of these cases. Finkelstein and Escherich and his pupils have confirmed these results, and have in addition presented the view that these diarrhœas are infectious, and may be caused by bacteria of the coli group. The bacteria may be introduced from without, or the coli organism in the gut under certain conditions may become virulent. With reference to their origin, these cases may be considered as bearing a relationship to cases of true dysentery, from which with our present imperfect knowledge it is not always possible to distinguish them.

**Morbid Anatomy.**—The mucous membrane is hyperæmic and swollen; in cases of long duration the mucosa is infiltrated with small round cells. The follicles of the gut are enlarged and elevated above the surface of the mucous membrane. The Peyer's patches are enlarged and surrounded by a zone of hyperæmia. The villi show desquamated epithelium and infiltration of the walls with small round cells. The follicles are swollen, and at the surface may



burst and present follicular ulcers. The epithelium of the gut may be lacking in places.

**Symptoms.**—In the beginning there are fever and slight vomiting. The movements are fluid, greenish, and have a disagreeable odor, contain mucus, and are streaked with blood. They may number ten or twelve in twenty-four hours. Straining at times accompanies the movement. As a rule the infant is pale and prostrated. The character of the movements is unchanged for from a few days to two or three weeks, when improvement begins and recovery ensues. On the other hand, in protracted cases the infant may develop a bronchopneumonia in one or both lungs, but may even then recover under good management. The picture thus resembles that of a mild dysentery, but the subjects are younger, and there is in a number of cases a history of antecedent intestinal disturbance of extensive duration.

The **treatment** should be carried out on the same lines as in acute gastro-enteritis. Caution should be exercised in returning to a diet composed exclusively of milk. While in true dysentery in older children I advise the administration of milk sterilized in some form, in younger infants such a procedure would be unwise. I keep these infants on a diet devoid of milk, such as beef-juice and barley-water, albumin-water or solution of acorn-cocoa, as long as possible. As the character of the movements improves the infants are put on a dilution of albumin-water and milk or cocoa and milk, or, what is far preferable, dextrinized gruel and milk. The amount of milk in the dextrinized mixture is gradually increased until the quantities appropriate to the age of the infant are given.

## DYSENTERY.

(*Illeocolitis*; *Colitis Contagiosa*; *Coli Colitis*; *Enteritis Follicularis*; *Enterocolitis*.)

Dysentery is an acute infectious diarrhœal affection of the intestine. In the United States it occurs both sporadically and in localized epidemics. It is endemic in the tropics, where the etiology is somewhat different from that in our climate. The protozoon infection (amœbic) seems, according to Kartullis, to be characteristic of the tropical form. Although amœbic dysentery is occasionally seen here sporadically and in cases of persons recently returned from the tropics, it is not the form which commonly occurs in infants and children. The form to which these patients are liable is seen during July, August, and September, and late in the autumn. It may affect nurslings who are fed artificially, but most often occurs in children who are on a mixed diet. Escherich has described epidemics of limited character in private families and hospitals. I have met this

form of dysentery in sporadic cases or small local outbreaks, and have also seen outbreaks at seaside resorts among children of from two to four years of age who had partaken of drinking-water which had been rendered unfit for use by contamination.

**Etiology.**—The essential cause of dysentery or ileocolitis is now recognized to be bacterial. It is due in certain cases to the introduction of bacteria of the coli group into the gut from without (Escherich). Maggiora, Celli, and others have described coli bacteria in the stools in epidemic dysentery. These authors have shown that these bacilli, which resemble the *Bacterium coli* of Escherich, may cause hemorrhagic colitis in lower animals. French writers think that the coli group existent in the gut may under abnormal conditions of intestinal disturbance assume a virulence not normal to them. Escherich, on the other hand, has endeavored to show that bacteria of the coli group, if introduced into the gut from without, either in the drinking-water or in food, may become very virulent. Among the other bacteria which have been found in isolated cases are forms of streptococci. These were isolated in a very severe case of sporadic dysentery in my hospital service.

**Morbid Anatomy.**—Dysentery may affect different sections of the gut at the same time, the rectal or sigmoid flexure alone, the ascending colon, the transverse or the descending colon only. In rare cases the disease may pass beyond the ileocaecal valve and involve the lower part of the ileum. There are two forms which may be present separately or simultaneously in the same gut—the catarrhal and the necrotic form.

In the milder catarrhal form of dysentery the mucous membrane is hyperæmic and swollen, and the summits of the intestinal folds are studded with hemorrhages in small foci or streaks. The sub-mucosa is infiltrated with small round cells and the vessels filled with blood. The epithelium of the follicles is swollen and proliferated, and there is infiltration of the surrounding connective tissue with round cells. In severe forms the surface of the mucous membrane is covered with mucus containing leucocytes and blood-cells. The follicles are elevated above the surface. In other cases the intestine is studded with ulcerations which mark the necrotic follicles. The ulcerations reach to the muscularis mucosæ. If the process extends to the small gut, the Peyer's patches are swollen and surrounded by a hyperæmic zone.

If the disease has advanced to the necrotic stage, the mucosa is thickened and infiltrated with round cells. There are areas of loss of tissue which extend deep to the muscular coat (gangrene). The mucous membrane is covered with a grayish exudate of a pseudo-membranous character. In severe cases large areas of the mucous membrane may necrose and be cast off. The necrotic areas show an abundant invasion of bacteria of the coli type, in scattered masses









tis. Some authors have recorded arthritis as a complication; as a rule it retrogrades and recovery takes place.

The **prognosis** varies with the severity of the case. The mortality ranges from 30 to 40 per cent. The croupous or necrotic cases are very fatal. With good management the mild cases give a favorable prognosis. The severity of the infection and the prevalence of an epidemic will influence the course of the affection.

**Treatment.—Prophylaxis.**—The movements are not only infectious, but may also communicate the disease to others if a particle is introduced into the gut. The hands of the patient and his body should be kept scrupulously clean to avoid reinfection. The movements should be disinfected in the same manner as those of a patient suffering with typhoid fever. The hands of the nurse should be scrupulously cleansed and washed in an antiseptic solution.

The patient is given a cathartic, preferably castor oil, as the initial step of treatment. In this way all irritating food particles and residual feces are cleared from the gut. All food, even milk, is withheld at first. The patient for the first twenty-four hours is given a solution of egg-albumin, acorn-cocoa, beef-juice broths, or expressed beef-juice and barley-water in equal parts. The following are the lines along which the later management of these cases should proceed:

a. An absolutely non-irritating and easily assimilable food is given.

b. The pain and tenderness are relieved with drugs, the diarrhœa being also partially controlled in this manner.

c. The rectum is irrigated.

d. After a day or two, during which the patient has been fed upon albumin-water, expressed beef-juice, and barley-water or acorn-cocoa solutions, sterilized or pasteurized milk is substituted. In these cases, as in typhoid fever, the patients are given during twenty-four hours, two or more quarts of milk sterilized at 212° F. (100° C.) or pasteurized at 164° F. (73° C.). I wait until the severely acute symptoms have subsided before placing these patients on a milk diet. At best, milk leaves a large residue in the gut, and in the acute stage of the disease the coagulum may in a mechanical way irritate the acutely inflamed walls. Pasteurized and sterilized milk is well borne in the later stages of the affection. Milk in a raw state, no matter how good, will sometimes tend to aggravate the acute symptoms. Pain and tenesmus are relieved by the exhibition of Dover's powder, grains  $\frac{1}{2}$  to  $\frac{1}{4}$  (0.03 to 0.12), every two hours according to the age of the infant or child. Codeine sulphate, grain  $\frac{1}{8}$  to  $\frac{1}{4}$  (0.01 to 0.015), according to the age of the patient, is preferable to morphine or tincture of opium. The administration of powdered ipecac-

uanha will be found very useful in certain cases. In others the vomiting rather interferes with its administration; grains j to ij or iij (0.06 to 0.12 or 0.2) every two or three hours are indicated. It may be combined with bismuth subcarbonate, grain v (0.3) every three hours.

In older children this mode of treatment has lately given good results. I have had no experience with the administration of lead salts. In the acute cases the internal administration of preparations, such as tannigen, is irritating.

Rectal enemata should be employed with care in the treatment of colitis or dysentery. Unless caution is exercised, their use is in many cases followed by an exacerbation or perpetuation of symptoms. The most useful form of enema is the warm ( $108^{\circ}$ – $110^{\circ}$  F.,  $42.2^{\circ}$ – $43.3^{\circ}$  C.) saline (Cantani) solution. Fully a quart of fluid is allowed to flow into the gut. The greater part of it returns, but I believe that if a portion of this solution is retained it acts in the manner of enteroclysis and supports the patient. These enemata are given three times in the twenty-four hours, for a day or two; they are subsequently given twice a day, and finally, as the symptoms subside, only once a day. I have never been able to convince myself that silver nitrate (1 : 1000) or tannic acid added to the enemata is of value. On the contrary, I believe that in cases in the acute stage these medicated enemata are distinctly irritating. In the later stages of the disease, small quantities of fluid blood are passed with the fecal movements, tenesmus being present; small enemata of silver nitrate (1 : 1000) given low down twice daily cause cessation of the bleeding which is due to the presence of ulcers low down in the rectum. In the subacute stage, the enemata will often be followed by an exacerbation of bloody mucous passages. Under these conditions it is well to discontinue the enemata and to watch the results of the suspension of local treatment.

### AMŒBIC DYSENTERY.

Amœbic dysentery is not, strictly speaking, a disease of infancy and childhood. It is caused by the *Amœbæ coli* of Lösch. Of 35 cases reported by Harris, 4 were under ten years of age. Amberg has recently published 5 additional cases. The etiological factor is the *Amœbæ coli*, which are found in large numbers in the movements. With the amœba, Charcot-Leyden crystals are found in most cases. The cases published by Amberg were of a mild type, and seemed in no way to differ in symptomatology from the form of the disease seen in the adult subject. There were diarrhœa of a bloody character, tenesmus, and in some cases fever and prostration. As many as from



four to six movements containing blood and mucus, and microscopically eosinophile cells, were passed in twenty-four hours.

The diagnosis is made from the presence of the amœbæ in the movements. Bloody passages containing Charcot-Leyden crystals should cause the physician to entertain a suspicion of the presence of this affection.

Other amœbæ, such as the *Monocercomonas hominis* (Grassi), have been found in the movements of infants suffering from diarrhœa. Epstein describes an epidemic of diarrhœa in which the *monocercomonas* abounded in the movements. He thinks that in this epidemic the diarrhœa was caused by well-water which contained the amœbæ. I have found the *Monocercomonas hominis* in the movements of infants who were suffering from diarrhœa, but also of those whose bowels were not in an abnormal condition. The rôle of the *monocercomonas* as an etiological factor in the causation of these diarrhœas is not understood. It is doubtful whether they have any causal connection with the diarrhœa.

## CONSTIPATION

(including the So-called Mucous Colitis or Mucous Disease).

Of all the conditions within the domain of pediatrics, constipation is the most difficult of treatment. It is not always possible to fix upon the cause of a constipated habit in infants. Infants at the breast may be constipated from birth and continue this habit through childhood, although normal in other respects. Sometimes the mother is of constipated habit. Slight rachitis may be present in particular cases. It is reasonable to conclude that some substance necessary to the normal action of the gut is lacking in the milk. After a time fissuration of the anus is developed and becomes a perpetuating cause of the constipation.

Rachitis, when marked, is associated with constipation in a large proportion of cases. The whole muscular apparatus lacks tone, and it is not surprising that both the muscle and the glandular apparatus of the gut lack power to perform the functions which are necessary to the maintenance of a normal state of the intestinal contents.

Hereditary influence has been named as a cause. I have met many cases of constipation in breast-fed and bottle-fed infants whose parents suffered from a similar condition of the gut.

Incorrect feeding is certainly a frequent cause of constipation in artificially fed infants and children. Some children who partake of boiled, sterilized, or pasteurized milk, become obstinately constipated, the condition being especially marked if the milk is subjected

to a temperature above 165° F. (73.5° C.). If there is a diminished quantity of fat in the milk, constipation will sometimes result. In older children a deficiency of certain articles of diet will cause constipation.

The rôle of fissure and eczema of the anus in causing and perpetuating constipation has been mentioned. In older children lack of exercise, nervous temperament, and lack of correct habits of evacuation of the bowel will cause constipation.

An account of the congenital anomalies and malformations of the gut and rectum as a cause of constipation does not lie within the scope of this section.

**Symptoms.**—One can scarcely speak of the symptoms of a condition which is itself a symptom. There are, however, certain features of the movements of constipated infants and children which are of importance.

**Movements.**—The movements of an infant suffering from constipation may be hard and formed, or may be unformed but dry. The movements in other cases consist almost entirely of marble-like masses resembling those seen in lower animals. Infants who are constipated do not have a movement unaided. In passing the movement they have pain, due in some cases to a fissuration and stretching of the anus by the hard masses. In other cases the fissures bleed at every movement. This bleeding, with slight prolapse of the gut during the movement, often creates the impression that the infant is suffering from a condition resembling hemorrhoids. Many movements are dry, others contain shreds and large masses of mucus or pseudomembranous structures. These masses are composed principally of mucus, and are not true membranes. The amount of mucus varies. The movements may be coated externally with the mucus or it may be passed in separate masses.

Frequently, infants and children do not appear to suffer from any ill effects as the result of constipation, but sometimes, and especially in children from the fourth to the sixth year, constipation is accompanied by crises of vomiting which occur at irregular intervals and are indicative of a species of intestinal toxæmia. Eustace Smith has described a similar condition under the heading of mucous colitis. It is probable that the mucous colitis is a result of the constipation rather than a primary condition. The attacks of intestinal toxæmia in constipated children begin with vomiting and dizziness. For a day or two prior to the attack the patients are noticed to be pale and listless; on awakening in the morning they complain of vertigo, nauseated feelings, and begin to vomit. At first some article of food ingested on the previous day is vomited. The vomiting persists even when the patient is in the recumbent posture. Anything taken into the stomach may be vomited—even water. After a time the vomited matter contains bile-pigment. The stomach may for a time tolerate



small quantities of fluid. These collect in the stomach and are finally vomited, the quantity vomited being equal to that taken into the stomach. The pulse is rapid and bounding; the cardiac impulse is forcible, resembling that seen in cardiac hypertrophy. The temperature may be elevated a degree above the normal, but rapidly becomes normal. In some cases there is pain about the umbilicus; there is no distention of the abdomen. The face is pale and anxious, and the patient appears greatly prostrated, as if suffering from a severe illness. The urine contains albumin; the urea is diminished, and at first there may be numerous hyaline and epithelial casts, which disappear after the subsidence of acute symptoms. The urine also contains ammonium urates. The vomiting subsides if the stomach is given rest, and in two days the patient appears to have made a recovery. During this time there is no movement from the bowel except with the aid of cathartics and enemata, but by these means good movement may be obtained, showing that there is no obstruction of the gut. Attacks are apt to recur. After the attack there is mucus in large quantities in the feces. It gradually diminishes in quantity, and finally under correct treatment disappears, but may reappear at intervals in varying quantity.

The **treatment** of constipation is dietetic, medicinal, and surgical.

If the infants who are constipated are fed at the breast, the mother's bowels should be regulated, and she should take regular exercise. In many cases the taking of nutritious diet by the mother will cause the milk to become changed in constitution and more abundant. In some cases, on the theory that the mother's milk is deficient in fats, the infants are given cream at regular intervals before nursing (Biedert, Holt). A teaspoonful of cream containing 16 per cent. of fat is given three or four times daily before nursing, with the result that in a day or two the bowels become regular. Sometimes infants under this treatment vomit or have diarrhoeal movements, especially in the summer. In other cases the infants continue constipated in spite of the administration of cream, and must be treated with drugs and massage. If artificially fed children are constipated, the heating of the milk should be stopped. If for some reason milk must be pasteurized or sterilized, the time of heating should be reduced to a minimum. Constipated infants may be fed on raw milk if the milk is fresh and carefully kept. The formula should contain sufficient fat to make the diet nutritious, but the fat should not form more than 4 per cent. of the mixture. As a rule, artificially fed infants do well on a smaller quantity of fat than the average breast-fed infant. Thus 2.5 to 3 per cent. of fat meet the requirements of most infants. If they are constipated, the fats are raised to 4 per cent. This proportion should not be increased, since there is danger of disturbing the functions of the

gut to such an extent as to give rise to conditions more serious than the constipation.

Children from the sixteenth month to the second year who suffer from constipation should be gradually weaned to a mixed diet. In many cases this procedure will regulate the bowels. The children should be given green vegetables, such as peas and spinach, in the form of a *purée*. The diet should include cereals of the various varieties, especially wheatena, oatmeal, granum, and rusk (Zwieback). The milk should be given raw with a moderate mixture of cream. Fruit, such as oranges, raw apples, and pears, is also given in moderation. If the constipation cannot be remedied by these measures, recourse is had to medicinal treatment.

**Cathartics.**—At best, cathartics are a makeshift. Some older children will do well with a small dose, grain  $\frac{1}{150}$  (0.0004), of strychnine once a day, and a simple cathartic, such as the aromatic fluid extract of cascara, twice or three times a week. A child two years of age may be given ℥ xx to xxx (1.0 to 2.0) once a day. The preparations of rhubarb are useful, but do not give uniformly satisfactory results. The mercurial cathartics are available only once a week in the majority of cases. We are thus reduced to the necessity of giving suppositories or enemata. With very young infants a small cylindrical piece of soap inserted with oil into the rectum once a day will be effective. With older children the glycerin suppository given every other day is very useful.

**Enemata.**—In many cases it is necessary to give enemata: to younger infants they are given once a day; to older children affected with the form of constipation occurring in connection with mucous colitis, an enema is given twice a week. The diet in these cases of mucous colitis should be carefully regulated; the movements are inspected, and articles of diet which are observed to pass undigested through the gut are avoided. When the child becomes pale and listless a brisk cathartic aided by a large high enema is given. In this way the attack of vomiting may be avoided. In cases of mucous colitis accompanied with crises of vomiting the children are kept in bed at the time of the crisis. No food of any kind, not even fluids, is given by the stomach. High rectal enemata are given twice daily. Somatose in solution is given by rectum, an ounce (30.0) three times daily. If vomiting persists, codeine is given in doses of grain  $\frac{1}{8}$  (0.01) by mouth. After twenty-four hours the administration of milk by mouth is begun cautiously. The diet list is gradually augmented, one article of diet being added daily until the child is taking a moderate fluid diet. When the stomach has become more tolerant a brisk saline cathartic, preferably Carlsbad salts, is given in milk. The patient is then gradually advanced to a full diet. In these cases it is of the utmost importance to discover what articles of diet agree with the patient. This can only be done by trying



each article in succession and inspecting the movements with a view to ascertaining the amount of residue. In convalescence the daily enemata are continued. These patients do well with some cathartic, preferably cascara, twice a week. Rectal enemas given twice a week should be continued for some length of time. Acidi muriatic. dil., ℥ ij (0.12), and pepsin, grains ij (0.12), t. i. d., will greatly aid stomach digestion in older children if there is pain after eating.

**Massage.**—Massage of the abdomen gives very unsatisfactory results. Gymnastics or calisthenic exercises in the morning after a bath are useful in some cases.

Useful formulæ are the following :

1. Pulv. glycyrrhizæ comp. . . . . ʒss to ʒj (2.0 to 4.0) as necessary.
2. Infus. sennæ comp. . . . . ʒj-ʒij (4.0-8.0) as necessary.
3. Podophyllin . . . . . gr ij (0.12).  
    Syr. rhei arom. . . . . ʒij (60.0).  
    Sig. ʒj (4.0) pro dosi.

The bitter waters may be given to older children as to adults.

**Surgical treatment** is directed toward remedying any local condition which may be present, such as fissure or spasm of the anus.

## ACUTE INTESTINAL OBSTRUCTION.

(*Intussusception.*)

Intussusception, according to Treves, is the prolapse of one part of the intestine into the lumen of an immediately adjoining part. It causes more than one-third of all the varieties of obstruction of the gut.

**Varieties.**—Invagination of the gut may take place in any part, from the duodenum to the rectum. There are the following forms :

The **enteric form**, which may involve any part of the small intestine, but which most commonly involves the lower part of the jejunum or the ileum.

The **colic form**, which may involve any portion of the colon.

The **ileocæcal**, which is the most common form.

In the ileocæcal variety the ileum and cæcum pass into the colon, the valve preceding and forming the apex of the intussusception. In the ileocolic form, the valve remains stationary and the ileum passes into the colon. In the latter form there is an invagination of the cæcum and colon, of a secondary character.

**Etiology.**—Nothnagel demonstrated that intussusception is caused by irregular muscular action in the wall of the gut; in acute intussusception this is of a spasmodic character. In 50 per cent. of the cases little is known of the exciting cause.

Diarrhœa, the various forms of enteritis, polypi, and diverticula, improper food, traumatism, and exposure to cold, have all been regarded as exciting causes. Typhoid fever and pertussis have been complicated or followed by intussusception. I have recently seen a case following typhoid fever in a boy three years old.

Meckel's diverticulum and the appendix have been the cause and seat of intussusception. In the latter case the inverted appendix caused ileocæcal intussusception.

**Frequency.**—Intussusception is more common in males than in females. The disproportion diminishes after the first year of life. Fifty per cent. of all the cases occur before the tenth year, and chiefly in individuals who are not in good physical condition (Treves). In the cases that I have seen, the infants were delicate, the child being robust in only one case.

The youngest case I have met was five and a half months of age. This infant was breast fed, had suffered with colic, and had had green movements from birth; there was an ileocæcal invagination eight inches in length.

**Symptoms.**—The onset is sudden in 75 per cent. of the cases; in the colic and rectal varieties it may be gradual. In many cases the disease makes its appearance while the infant is nursing or during sleep. The patient, being attacked with pain, suddenly awakes from sleep with a cry and begins to vomit; on the same day or the following day a bloody movement appears, the amount of feces being small. In a few cases there are no fecal evacuations. If the case is progressive, the pain returns in paroxysms, the hemorrhagic movements are repeated, and the vomiting keeps pace with the increase of the obstruction. The general condition of the patient grows worse; apathy and collapse ensue. I have seen cases begin with a mild diarrhœa; the pain suddenly appears, and also the hemorrhages from the bowel, the infant at once going into collapse. There is apathy, from which it is difficult to rouse the patient. If the case continues to progress, the movements become frequent, exhaustion increases, and finally death from asthenia results. The pain is great at the onset, usually reaches its maximum intensity within a short time, and then gradually subsides. It is of a paroxysmal character and is colicky during the advance of the invagination; as adhesion takes place or gangrene occurs it diminishes. The intervals between the paroxysms of pain are at first of considerable length; later they become shorter. The pain is most severe in the ileocæcal form, and is in all forms caused by irregular intestinal peristalsis.

**Vomiting** is not so prominent a symptom as in other forms of intestinal obstruction (Treves). In 75 per cent. of the cases it comes on early with or directly after the pain. It may not recur for hours. In a child taken with sudden pain of a colicky character, vomiting, and bloody stools, the vomiting recurred only twice within



twenty-four hours. It is apt to be less violent as long as there is not complete obstruction of the gut; in other words, it is more marked in those cases in which no feces pass through the gut. As long as the pain recurs in paroxysms (progression of the intussusceptum) the vomiting is not apt to be marked. The vomited matter is composed of the stomach contents and is biliary; stercoraceous vomiting was found late in only 25 per cent. of Leichtenstern's cases; Gibson also found it to be rare and late. If stercoraceous vomiting was present, it appeared from the fourth to the seventh or to the fourteenth day. In the case referred to, in the infant of five and one-half months, it appeared during the first twelve hours of the disease.

The condition of the bowel is important. It is generally stated that constipation occurs from the outset; this is not universally true. Cases in which constipation exists throughout, that is to say, in which no feces whatever are passed, are not common, and form only 30 per cent. of the total number. Diarrhœa is the common condition at the outset; as the obstruction increases, the amount of feces in the stools diminishes, and finally only mucus and blood are passed.

The most important symptom in connection with the bowels is hemorrhage. Hemorrhage from the bowel, in connection with pain and other abdominal symptoms, is considered by Gibson as pathognomonic. It was present in 80 per cent. of the cases tabulated by Leichtenstern. As a rule it is considerable. It is said by Treves to have been in some cases so great as to cause death. The blood and feces have a cadaveric odor, which however is not always, as some writers affirm, a sign of gangrene. I have perceived this odor in an intussusception which operation showed not to be the seat of gangrene. It is caused by decomposition of the blood in the gut.

The temperature is normal, slightly subnormal, or slightly elevated. There may be a slight elevation of temperature without peritonitis. The quantity of urine may as in other forms of intestinal obstruction be diminished.

**Tenesmus** is present in 55 per cent. of the cases; it depends more or less on the presence of the intussusception in the rectum. It is usually an early symptom in the rectal form, and is more common in the ileocæcal variety than in the enteric.

The abdomen is not at first distended; it may, on the contrary, be retracted; if tympanites occurs at all, it does so late and in the presence of a general peritonitis. Palpation of the abdomen is at first well borne, but after a time there is sensitiveness.

A **tumor** felt through the abdominal wall or in the rectum is of the greatest value in the diagnosis. It cannot be felt if the intussusception is in the hepatic or splenic flexure of the colon. It is variable in distinctness, and is most frequently felt in the region of the descending colon or of the sigmoid flexure. It is hard and resistant,

and rarely more than six inches long. It is often said to be sausage-shaped, but the statement is misleading. The tumor is rarely felt in the ileocaecal region, for the reason that the intussusception in this locality is small, and is that of a small gut inside of a large one. In one-third of the cases the rectum, if examined, shows the presence of the intussusceptum. The rectal tumor is commonly found in children, because in them the colon is mobile. The gut may reach the anus as early as the second day, the average time being the seventh day. It may protrude from the anus from three to eight inches, and may be in a gangrenous state; under these conditions it has been mistaken for a polypus or hemorrhoid.

**Prognosis.**—As regards duration, there are three varieties of intussusception—the ultra acute, the acute, and the subacute. The ultra acute cases are exceedingly rare. Leichtenstern found only 5 of this form in a total of 7269 cases; 4 of the 5 occurred in infants less than a year old. All were fatal.

The rate of mortality in intussusception, excluding the ultra acute forms, varies as given in the statements of different authors. Gibson's statistics place the mortality at 53 per cent. It varies with the age of the patient, the duration of the disease before operating, and the success in reducing the intussusception. Intussusception is extremely fatal in infants under the first year. Thus according to Treves, the mortality under one year of age is 80 per cent. On the other hand, if we study the cases as Gibson has done, we find that the cases operated on during the first day of the disease had a mortality of 41 per cent.; those on the fourth day, 72 per cent. The reducible cases showed a mortality of 38 per cent.; the irreducible, of 88 per cent.

**Diagnosis.**—From the studies made by Gibson, it may be seen that, in children, a bloody discharge with abdominal pain of a paroxysmal nature is almost pathognomonic of intussusception. Fecal vomiting is of very little value as a diagnostic sign. It is very infrequent, and is in any case present only late in the disease, when occlusion of the gut has occurred. If enteritis exists in a young infant, it is often difficult in the absence of any abdominal or rectal tumor to make a diagnosis. The course of the case will guide the physician. In dysentery the hemorrhage from the bowel is not great; it is composed of blood-tinged mucus. Appendicitis has been mistaken for intussusception. It frequently occurs with it, and thus obscures the picture. Peritonitis can hardly be mistaken for intussusception. In peritonitis the pain is continuous and there is tympanites, but no bloody discharge. Tuberculous peritonitis is sometimes mistaken for intussusception. In tuberculous peritonitis the symptoms are not progressive, and also there is not likely to be a bloody discharge.

The case following typhoid fever, to which I referred, simulated



a hemorrhage from a typhoidal ulcer. A careful examination under an anæsthetic cleared up the case. In complete relaxation under anæsthesia, a tumor could be felt in the cæcal region of the ascending colon. The result of examination was verified by operation. In all doubtful cases in which the restlessness of the child interferes with a careful examination an anæsthetic should be given. There is a characteristic condition which in some cases can be detected by examination. As the finger is inserted into the anus the rectum is felt to be inflated. This is due to traction on the gut by the invagination. I have found this inflated state of the rectum in two infants suffering from intussusception.

**Spontaneous Cure.**—There is little doubt of the possibility of spontaneous recovery in invagination; such cases have been recorded by competent observers. Henoch has seen typical intussusception retrograde and the patient recover. There is another mode of recovery which occurs in cases of irreducible intussusception: the intussusceptum sloughs off and is passed per anum. This occurred in 43 per cent. of the unrelieved cases (Leichtenstern), but in 40 per cent. of these the patient succumbed to general sepsis with or without peritonitis or to subsequent obstruction of the gut from swelling after the gangrenous portion was passed. Henoch reported a case of this kind.

**Treatment.**—The diagnosis of intussusception once made, the case is one for surgical interference. The sooner surgical treatment is begun, the better the chances of recovery. Injections of air, gas under pressure, and enemata of water and oil have been tried, with some measure of success. Their use, however, delays the radical treatment, and apparent improvement frequently gives way to an exacerbation of symptoms. Surgical aid then comes too late. The objections to the treatment by injection are as follows: the gut is viable in these cases, and is liable to be ruptured by injection of gas or air under pressure; an enema of water under only four feet of pressure has been known to produce this result. Snow published a case in which an injection of oil was made; post mortem the oil was found above the point of obstruction. The enema may thus pass through the lumen of the gut without relieving the intussusception. Enemata should be given, if at all, during the first forty-eight hours, and should be allowed to flow into the rectum under very low pressure. The amount of fluid varies; certainly not more than a quart should be given. The fluid, a saline solution at 100° F. (37.7° C.), is allowed to remain in the rectum for ten minutes, the patient being under an anæsthetic. A Davidson syringe should not be used. The ordinary bag irrigator is best for this purpose. If one enema fails and the diagnosis is certain, there should be no delay in seeking surgical assistance.

## APPENDICITIS.

(*Perityphlitis; Paratyphlitis.*)

**Anatomical Peculiarities.**—Vallée examined the appendix in 100 infants and children post mortem. He found that in fully 75 per cent. the cæcum is situated above the anterior superior spine, on the right side, a position higher than that occupied in the adult. It is above the plane of the anterior superior spine of the ileum, is almost 5 centimetres long, and has a general longitudinal ascending or descending direction. In one case the appendix was situated entirely to the left of the median line, there being no transposition of the other viscera. Knowledge of these facts is of importance in the examination for the appendix in conditions of disease. I have frequently succeeded in palpating the normal appendix at one side of the cæcum. It is felt as a cylindrical body having the diameter of a quill.

### Acute Appendicitis.

**Frequency.**—Although the statistics showing the frequency of appendicitis in infancy and childhood vary with the number of cases collected by each author, the combined statistics of Matterstock, Fitz, Sonnenburg, and Nothnagel, show that the disease is not very frequent before the tenth year. Only 8 per cent. of the cases occur at this age. It may occur in early infancy. Savage records a case in an infant two months old; Demme also records a case in a very young infant.

The literature shows occasional cases at all periods of infancy. Among the cases collected and tabulated from the service of my colleagues, Gerster and Lillienthal, at the Mount Sinai Hospital, there is one of an infant one year of age. Of 50 cases of appendicitis in children taken from the service of these surgeons, 1 occurred in an infant one year of age, 17 from the third to the sixth year, and 32 from the sixth to the tenth year of life. Thus in a statistical collection of cases occurring in children, only one-third occurred before the sixth year of life.

The **forms of the disease** are the same as in the adult subject. The perforative form seems to be the most common among children. Thus of 50 cases coming to the hospital for operation, 31 were perforative with or without abscess, 9 were of the gangrenous variety, and 6 of the catarrhal form. It will thus be seen that in children the tendency in this disease as in others, such as pleurisy, is toward suppuration and the formation of abscess.

The **symptoms** will vary with the variety, whether catarrhal, perforative, or gangrenous.

a. In the **catarrhal form** the patient is, after some indiscretion in diet, seized with colicky abdominal pain, vomiting, and some



fever. In other cases the children simply complain of pain which is not sufficiently severe to prevent their being up and about. The pain is not always located by the patient in the appendix. When the patients are in the recumbent posture, the right knee may be flexed and the thigh flexed on the abdomen; when they walk, they do so in a bent position, favoring the affected side. Physical examination reveals a localized resistance or tenderness in the right iliac fossa. In some cases there is distention of the cæcum with feces, in others I have felt the appendix and the cæcum matted together in a mass of the size of the index finger.

The pain is not always referred to the iliac fossa, but may be distinctly located around the umbilicus or over the lower part of the abdomen.

It may not always be possible to palpate the appendix, which may be behind the cæcum. Under such conditions no intumescence will be found. McBurney's point will be considered in the diagnosis.

The history of many of the catarrhal cases is one of recovery under careful treatment. The fever subsides or may never have been above  $101^{\circ}$  F. ( $38.3^{\circ}$  C.); the pain also subsides, and in from a few days to a week the patient is apparently well. Attacks of this kind may recur.

*b.* In the **perforative** or **suppurative** form the symptoms are more violent. In this form also the onset of the disease seems to date from some indiscretion in diet. The patient is seized with sudden sharp pains in the abdomen, accompanied by vomiting, fever, and rapidity of pulse. The pain is located either in the upper or the lower part of the abdomen, or in a few cases in the right iliac fossa. After one or two attacks of vomiting this symptom may subside and not recur until the second or third day, when perforation occurs. Tympanites occurs early and may set in after the second day of the disease. The pain and tympanites cause an increase in the respiratory movements, which are shallow. The patients lie in the recumbent posture. The tympanites, if the perforation is extensive and there is general peritonitis, causes, as in all forms of perforation, a disappearance of the liver dulness. The pulse is at first rapid and thready, and quickly mounts above 120 after perforation has occurred. The prostration is great, and in some cases of a septic type jaundice is present.

*c.* In the **gangrenous** form the symptoms are very similar to those of the perforative form, but are very much intensified. It is not possible to tell from the symptoms whether the process is gangrenous, simply perforative, or catarrhal followed by abscess.

**Course.**—In both the perforative and the gangrenous cases in children as in the adult, localized adhesions may form with a small or large collection of pus or several foci of pus. In other cases a

general peritonitis follows the perforation. In children, as in adults, the moment of perforation is followed by a temporary fall in the temperature and a cessation in the pain and vomiting, the pulse, however, continuing rapid. The lull, however, is of short duration, and is quickly followed by an increase in the severity of the symptoms.

**Diagnosis.**—The above outline gives very little idea of the great and sometimes insurmountable difficulties of diagnosis of appendicitis in young children. To guard against error, a very careful routine should be followed. The patient is completely undressed and lies in the recumbent posture, the shoulders being slightly raised. The physician should stand or sit at the patient's right. The contour of the abdomen is noted. If it is normal and not distended, there is probably no peritonitis. The abdomen is very gently palpated in different places to ascertain if there is distributed or localized tenderness.

FIG. 93.



Method of examination of the region of the appendix vermiformis.

The left palm is then placed underneath the right loin, and with the palmar surface of the fingers of the right hand the region of the appendix is gently palpated (Fig. 93). Superficial palpation is practised at first. The hand is then depressed deeper into the iliac fossa in search of resistance or tumor. The intensity of the pain caused by manipulation is carefully gauged by watching the face of the patient. The right iliac region having been carefully palpated, rectal exploration should be made in all doubtful cases. This is necessary in the cases in which a general tympanites or general abdominal tenderness makes the diagnosis difficult. With the well-oiled index finger of the right hand the rectum is explored as high up as possible. In young children this can be done without causing pain if gentleness and caution are exercised. If children are very intractable, this method of examination cannot be carried out.



The following points are important in the diagnosis :

**Tympanites.**—If the abdomen is distended and there is general pain with increase of the number of respirations, there is probably peritonitis localized or diffuse. In the latter case there is disappearance of the liver dulness if the tympanites is extreme.

**Percussion** will sometimes, even in general peritonitis, give a localized dulness in the right iliac fossa. Localized pain and intumescence or a localized mass in the right iliac fossa are of great import.

**McBurney's point** is of less value in children than in the adult. In children, as will be seen from Vallée's work, the appendix is situated higher than in the adult, and McBurney's point is therefore too low for palpation. Some children complain of epigastric, others of umbilical pain, which is not so distinctly localized as in the adult.

The **fever** is of little value, there being nothing characteristic in the curve. The temperature may be normal or in severely septic cases slightly raised. After perforation, the temperature becomes subnormal, as it does in the adult.

**Appendicitis** in children may simulate **tuberculous peritonitis**. In the latter disease there is sometimes severe pain of the colicky variety. Tuberculous peritonitis and appendicitis may be coincident.

**Pain** in appendicitis resembles very closely that in gastro-enteritis and dysentery. Griffith has published 2 cases of appendicitis in children who had entero-colitis at the same time.

I have had one case in which a **perinephritic abscess** simulated an appendicitis. The contrary may also occur. Appendicular abscess may simulate a coxalgia with abscess. I have seen a few cases of typhoidal affection of the appendix which for a few days simulated an appendicitis very closely. Appendicitis with invagination of the appendix into the cæcum is a rare condition, as is also intussusception with appendicitis. In the typhoidal cases, a Widal reaction may be obtained, and will be of assistance in diagnosis. Care should be taken that a perforating typhoidal ulcer does not escape diagnosis. Intussusception will give the characteristic symptoms of that condition.

I have seen cases of **lobar pneumonia** of the lower lobe of the right lung, in which the pleuritic pain radiated down the right side into the iliac fossa. There were also epigastric pain and vomiting at the onset of the disease. The excessive rapidity of the respirations, the marked dyspnoea, and absence of tympanites and pain on deep pressure in the region of the appendix, led me to examine the lung.

**Prognosis.**—Of the 50 hospital cases which I have tabulated above, only 3 recovered without operation; they were of the

catarrhal variety. These figures give no accurate idea of the proportion of recoveries made under careful and conservative treatment in private practice.

The mortality in the cases operated upon was 35 per cent. The rate is not high considering that many cases came under the knife later than would have been the case in private practice. On the other hand, it should be remembered that the rate of mortality is also influenced by the nature of the infection and the power of resistance of the patient. Thus cases with a gangrenous appendix died although operated upon on the second day; others of the same kind recovered although the disease had lasted from four to seven days before operation. Some perforative cases died on the second or third day of the disease, while others recovered although operated upon from six to twelve days after the onset of symptoms. Gangrenous cases in this statistical table in children show a lower rate of mortality than those cases in which the appendix perforates, forms an abscess, and causes general peritonitis.

### Chronic Appendicitis.

This form of appendicitis occurs in older children. The cases are frequently mistaken for those of dyspepsia, and *vice versa*. The history is much the same as in the adult. A child otherwise in good health has attacks during which there is abdominal pain not of great severity, but which may last for a few hours and disappear, leaving the patient well. The pain is very rarely referred to the appendix; it is abdominal, the umbilical region being generally indicated as the seat of discomfort. The temperature may reach 100° F. (37.7° C.); the pulse in a child of eight years was 96 and regular. There is no vomiting and no prostration. The pain is sufficiently severe to make the patient wish to lie down; it is not excessive when the appendix is palpated. The bowels are regular. The cases may in the intervals between the attacks show a slight intumescence in the region of the appendix, but nothing is felt in the rectum. The signs in the interval may be very indefinite or quite distinct. The cæcum and appendix are felt to be matted together. Three cases in which there had been repeated attacks extending over a period of from one to two years, were operated upon for me by leading surgeons. The patients were girls between the ages of six and eight years. In each case there was evidence of a chronic catarrhal process. In one case the appendix contained a fecal calculus, in another there were constricting adhesions.

The **treatment** of both acute and chronic appendicitis in infants and children does not differ from that followed in the adult subject.



### RECTUM.

In infants a large portion of the rectum is situated in the abdominal cavity rather than in the pelvis. It has three curves—one lateral and two anteroposterior. The gut is nearly straight and occupies a more or less vertical position, hence the frequency of prolapse. The attachment of the rectum to the surrounding parts is not extended as high in children as in adults, hence the rectum is more liable to be pushed out. The rectum of the newborn infant may be divided into three parts. The first lies in front of the sacrum and ends at the lower end of the bone; the second is short, and in this respect differs from the adult gut, being also more vertical; the third portion is long, and extends downward and somewhat backward. The second portion being short, when the rectum is distended, the gut is straightened out and the whole rectum extends downward and backward (Symington). All these data are of importance in applying methods of therapy (enteroclysis, etc.) to this organ.

### Prolapsus Ani.

Prolapsus ani is a condition frequently met with in infants and children. It may amount only to an eversion of the mucous membrane. There is in some cases a complete descent of part of the rectum, which protrudes from the anus to the length of one or two inches.

The **etiology** of this condition is obscure. It evidently only occurs in cases in which the pelvic attachments of the lower bowel are lax. It is favored by anatomical conditions elsewhere mentioned. It is seen in children who are constipated, in those who suffer from diarrhoea, and also in those whose movements are not normal. Any abnormal condition in the neighboring organs, such as the bladder and urethra (stone), may cause excessive straining and consequent prolapse of the gut. A rectal polypus may cause prolapse.

**Symptoms.**—In some cases the only symptom is the appearance of a small quantity of mucus and blood on the diaper with each movement; in these cases the prolapse returns spontaneously. In other cases the bowel descends to the extent of one or two inches with the movement, and remains prolapsed. If a polypus of the lower part of the rectum is the cause of the prolapse, it is seen protruding from the prolapsed portion.

**Treatment.**—The first step is to replace the protruding gut. The gut is anointed with olive oil or vaseline and gently replaced with a towel. The movements are so regulated by diet and cathartics that the stools are passed without straining. Three times daily a suppository containing grains ij to iij (0.12 to 0.2) of tannic acid is placed in the lower bowel. While the movements are being passed

the patient is kept in the recumbent posture on a bedpan. This treatment is frequently successful. In other cases, the buttocks are drawn together by adhesive straps and the child is allowed to pass movements thus strapped. Cocaine and strychnine are used both in suppositories and hypodermically. The protruding portion is painted with cocaine. These measures have their failures and successes. The only satisfactory method is that first advised—of a strict diet, the recumbent posture at stool, and the astringent suppository. The Paquelin cautery is sometimes employed to cauterize the mucous membrane. The danger in this method is the substitution of a traumatic stricture of the anus for the comparatively harmless prolapse. Application of the pure stick of silver nitrate to the anus twice a week, has given good results. If a polypus of the rectum is the cause of the prolapse, the growth should be removed by surgical means.

#### **Fissure of the Anus.**

Fissure of the anus is seen in syphilitic infants, in those suffering from marked constipation, and in infants that have eczema of the anus. It may be the result of the repeated introduction of the hard nozzle of an enema syringe. The fissure may be so slight as to be only a line-like tearing of the mucous membrane, or may consist of a broad ulcer with a hard granulating base.

**Symptoms.**—As a rule, the infants are constipated. When a movement is passed, the infant cries and there is great pain. A few drops of blood are passed on the diaper.

**Diagnosis.**—The presence of a fissure of the anus sometimes escapes the notice of the physician. If there is a history of the above symptoms, the physician should place the infant on a table, grasp the buttocks with the palm of the hands and separate them forcibly with the thumb. The anus is thus everted, and if a fissure is present it will at once become apparent.

**Treatment.**—A small fissure is sometimes very successfully treated by regulating the bowels. It is touched with a 10 per cent. solution of silver nitrate once a day. In the severe cases silver applications will not avail; forcible dilatation of the rectum by means of the thumbs must be resorted to. This procedure not only cures the fissure, but is also an effectual remedy for the accompanying constipation.

#### **Spasm of the Anus.**

Cases of nervous spasm of the sphincter ani occur in infants. The infant is constipated and cries at each movement. There is no bleeding, nor does examination reveal any fissure, but only marked contracture of the anal opening. In these cases it is almost impossible in an examination to bring down the upper part of the anal gut.



The remedy is to regulate the bowels. If by this means success in overcoming the spasm is not attained, forcible dilatation is the only resource.

### Proctitis.

Apart from the membranous and catarrhal forms of proctitis, which occur with similar conditions of the intestine, the only form which is of interest is the gonorrhœal. This occurs as a complication of vulvovaginal gonorrhœal inflammation. In these cases the introduction of the gonococcus from the vagina into the gut has occurred through careless thermometry or the giving of enemata without previous cleansing of the parts. The disease is very painful and at the same time trying to the infant or child. With the discharge of pus from the anus there are tenesmus and a bloody discharge with the movements. The purulent discharge shows gonococci.

The **treatment** consists in the injection of protargol solutions, 2 per cent., at a temperature of 105° to 108° F. (40.5° to 42.5° C.), into the rectum twice daily. The bowels are regulated. Suppositories of tannin or tannigen are also of value and give great relief; one containing grains iij (0.18) is given per rectum twice daily. In the later stages it may be necessary to paint the lower bowel with a very weak solution (0.5 per cent.) of silver nitrate.

### Polypus of the Rectum.

Polypus of the rectum is not rare in childhood, but is not often seen in infancy. It occurs most frequently from the third to the seventh year of life. The polypi are adenomata. I have examined several, and have found them to have the structure described by Baginsky. They may be single or multiple, usually have a pedicle, but may be attached to the wall of the gut by a broad base. As a rule they are situated on the posterior wall of the rectum seven or eight centimetres above the anal ring, but may be on the anterior wall. In most cases the polypi exist here only, but I have seen them higher up in the gut, and in one case in a child of five years from whom several rectal polypi had previously been removed, I diagnosed a number in the descending colon. In this case laparotomy and incision of the gut proved the diagnosis to have been correct. The polypi may, if they become numerous, assume a malignant character; this is especially true of the growths with a large, broad intestinal base.

**Symptoms.**—The characteristic symptom is intermittent hemorrhages from the gut, which may be profuse. At times the outer surface of the movements is streaked with blood, the bowels being constipated or normal, with an occasional mucous diarrhœa. If the

polypus is low down, there is straining at stool with prolapsus of the gut. Many of the children thus affected are pale, have a pasty hue of the skin, and show evidences of lymphatism.

**Diagnosis.**—Bleeding from the bowel, in the absence of other symptoms, should at once suggest the necessity of digital exploration of the lower bowel. If a polypus is not found, a careful palpation of the abdomen made when the patient is fasting should be the next procedure. If the child is tractable and the abdomen soft, it may be possible to feel a tumor of the size of a hazelnut at one side of the umbilicus.

The **prognosis** is good; removal of the polypi is rarely followed by recurrence of symptoms, even in cases in which they are situated in the descending colon. If they are removable and not very numerous, the patient recovers.

**Treatment.**—If the polypus is low down and pedunculated, it may easily be snared with or without the aid of a rectal speculum, and crushed or ligated off. If it is high in the sigmoid flexure, the anus should be dilated and the growth reached by means of a speculum. In cases in which the growth is in the colon, laparotomy, enterotomy, and ligation are indicated.

### INTESTINAL PARASITES.

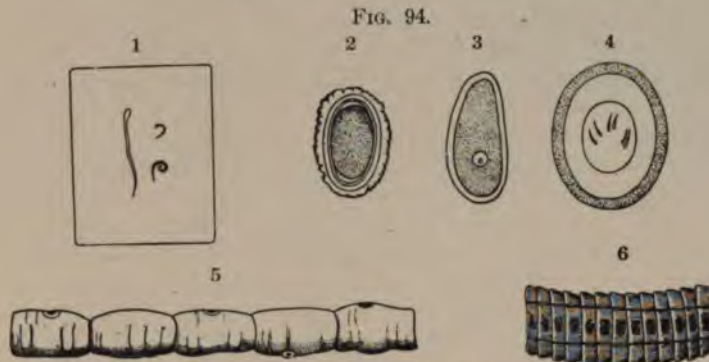
The most common parasites found in infants and children are the Nematoda, or round worms, and the Cestoda, or tapeworms. The round worm is smooth and light brown or reddish in color, the female being larger than the male. The eggs are found in the stools; they are from 0.05 to 0.06 mm. in diameter and are surrounded by an albuminous envelope. The worm is several inches long. *Oxyuris vermicularis* is about 1 cm. long, the male having a length of 4 mm. The eggs measure 0.05 mm. in their long diameter.

The tapeworms in mature state consist of rectangular segments. The head and neck are called the scolex; the segments, proglottides. The worms are hermaphrodites. The solium is sometimes several metres long. The head is of the size of a pin's head, with a projecting proboscis armed with hooklets. The eggs of the solium are ovoidal, 0.3 mm. in diameter. The *Tænia mediocanellata* has a more cuboidal head without hooklets (Fig. 94).

**Diagnosis.**—There are no symptoms which can be traced to the presence of these worms in the gut. If they increase in enormous numbers, they may cause symptoms of mechanical obstruction. Without the presence of the eggs or links of the worm, a diagnosis is not possible. Their presence is made known by the passage per anus of the links of such worms as the tapeworm. Round worms



may also pass out of the anus, or may be vomited if they gain access to the stomach. Thread worms may cause excessive pruritus, and may not be discovered external to the anus. In that case the feces should be carefully examined for the eggs of the worms.



1. *Oxyuris vermicularis*, pin worm, natural size.
2. Egg of *Ascaris lumbricoides*.
3. Egg of *Oxyuris vermicularis*, pin worm.
4. Egg of *Taenia solium*.
5. Proglottides or links of *Taenia solium*.
6. Proglottides of *Bothriocephalus latus*.

### Round Worms.

(*Ascarides Lumbricoides*.)

This parasite is found in the small gut ; it may invade the stomach or may pass downward into the rectum. Cases are recorded (Borger) in which it has passed into the bile-duct and caused abscess of the liver. There may be only one or many of these worms in the gut. Leuckart states that they may form large masses in the gut, and thus cause intestinal obstruction. They have been known to perforate the gut and cause peritonitis. The eggs are introduced into the gut through the medium of drinking-water, fruit, and vegetables. Epstein cultivated the eggs outside of the body and then introduced them into the gut, where they developed. The male worm is 250 mm. long, the female being longer.

The **symptoms** caused when these parasites have once gained access to the body are not characteristic. I have seen the worms passed or vomited by children apparently in normal condition.

The **treatment** consists in placing the patient on a milk diet. After a few days the following powder is administered two or three times daily :

Calômel,  
Santonin . . . . . 55 gr.  $\frac{1}{2}$  (0.016).

Santonin is sometimes administered in the form of pastiles, but is not more satisfactory than the above preparation.

**Oxyuris Vermicularis.**

(Pin Worm; Thread Worm.)

Brass showed that the habitat of these worms is the small intestine, whence they pass into the cæcum. The female worm lays its ova in the folds of the gut. They may pass into the stomach and thence into the mouth, but more frequently pass out of the anus into the vagina or into the prepuce and urethra. They exist in enormous numbers in the gut, are exceedingly small, and have the appearance of fibres of cotton fabric. They can be seen by spreading the nates apart. They are then found in the anus, or in female children in the fourchette. The principal symptom is intolerable pruritus, so intense as to deprive the children of sleep. This worm is found only in the human subject. It is conveyed from person to person through uncleanness. The larvæ adhere to the fingers, and thence are introduced into food-stuffs.

**Treatment.**—It is a very difficult task to dislodge these worms; injections by the rectum cannot reach those higher in the intestine. The plan which I have followed, and which gives relief, is to give daily enemata of quassia wood before bedtime:

Quassia wood (ground) . . . . . ʒj (31.0).  
Aque dest. . . . . Oj (500.0).  
Make an infusion and strain.

I have in addition utilized the prescription of santolin and calomel given above for the round worms.

Schmitz recommends the administration of naphthalin, grains j to iij (0.06 to 0.18), t. i. d., for a week, after which it is discontinued for a few days, and then given again.

**Tapeworm.**

(Tænia.)

Tænia are quite common in children, and have been found in the newly born infant (Müller and Armor). In these cases the eggs must have been deposited in the liquor amnii, and thus swallowed by the fetus. Numerous cases have been recorded of the presence of these worms in infants from the third to the twelfth month. They are most frequently found between the first and the third year. The varieties most commonly found in children are: Tænia solium, Tænia mediocanellata, Tænia elliptica, Bothriocephalus latus.

**Sources of Origin.**—**Tænia Elliptica.**—The lice of the house-dog and cat are introduced by the fingers of the children into their mouths, and thus gain access to the gut. There the larvæ of the tapeworm which they contain develop.

**Tænia Solium.**—The larvæ of this worm are found in badly cooked pork or beef.



**Tænia Mediocanellata.**—The larvæ of this worm are found in beef. *Bothriocephalus latus* is introduced by the ingestion of infected fish-food.

The larvæ of tapeworm may exist in the flesh of the hare, pigeon, pheasant, chicken, goose, or duck. Ice if made from infected water may be a means of introducing the larvæ in the body. It is thus not necessarily the meat-eating children who run the danger of swallowing the larvæ of tapeworm; milk if diluted with infected water may contain them.

**Symptoms.**—Tapeworms may exist for months or years in the body of a child without causing untoward symptoms. As many as three varieties of the worm have been found in the same child. The symptoms are not characteristic. The passage in the movements of the links of the tænia is the only positive evidence of their presence.

**Treatment.**—The only successful treatment for the expulsion of the tapeworm is that which consists in the administration of filix mas in some form. It should be freshly prepared and given in liberal doses: Ext. æth. filix mas, ℥xxx (2.0) to ʒj or ʒij (4.0 or 8.0), is made into an emulsion with gum tragacanth, and mixed with equal parts of castor oil. The administration of this mixture is preceded by a day or more of milk diet. The child is then given from half a drachm to a drachm (2.0 to 4.0) of the filix mas with castor oil in divided doses. The recumbent posture is maintained in case nausea should be experienced. The movements containing the worm are carefully washed through a sieve, and the smallest part of the worm sought for in order to see if the head has come away.

The patient should be given a drawing of the comparative size of the head and links of the worm, in order that the head may not be lost, or the physician may seek it himself.

#### LEADING AUTHORITIES REFERRED TO IN CHAPTER V.

- Amberg, S.: "A Contribution to Amoebic Dysentery," etc., Johns Hopkins Bull., December, 1901.  
 Borger, G.: "Ascaris Lumbricoides," etc., Arbeit. aus der k. univ. Kinderlinik, Munich, 1891.  
 Biedert, P.: Diätetische Behandlung der Verdauungsstörungen, Stuttgart, 1901.  
 Booker, Wm.: "A Bacteriological and Anatomical Study of the Summer Diarrhoeas," etc., Johns Hopkins Hospital Rep., vol. v.  
 Beck, Carl: "Appendicitis," Volkmann's Vorträge, No. 221.  
 Czerny and Keller: Ernährungstörungen, 1901.  
 Epstein, A.: "Beobachtungen über Monocercomonas," etc., Prager med. Wochen., 1893.  
 Escherich, T.: Streptokokken Enteritis, Jahrbuch. f. Kinderheilk., Bd. XLIX.  
 ——"Dysenterie," Centralbl. f. Bakt., 1899.  
 ——"Ätiol. der Primären ac. Magen Darm Krankheit," Wien. med. Wochen., 1900.  
 Filatow, N.: Darmkatarrh der Kinder, Vienna, 1893.  
 Gibson, C. L.: "One Thousand Operations for Acute Intestinal Obstruction," Annals of Surgery, 1900.

- Groppe*, F.: Fettsäure t. der Frauenmilch, etc., Volkmann, 1901.  
*Holmer*, G.: "Atrophie," *Jahrbuch. f. Kinderheilk.*, N. F., liii.  
 -- *Atrophien u. Schwinden*, Berlin, 1908.  
*Leubner*, R.: Die Parasiten des Menschen, Leipzig, 1894.  
*Morjau*, A. B.: *Gastro-enterites des Nourissons*, Paris, 1900.  
 -- *Traite de l'Alaitement*, Paris, 1899.  
*Moss*, J. L.: "Renal Complications of Acute Enteric Diseases," *Trans. Amer. Ped. Soc.*, 1899.  
*Nord*, L. H.: "Congenital Stenosis of the Pylorus," *Pediatrics*, 1901.  
*Hjorth*, M.: *Veter. Stoffwechsel Störungen*, Berlin, 1901.  
 Magen Capacitat, etc., *Bibliotheca Medica*, Stuttgart, 1898.  
*Prinkard*, E.: "Congenital Pyloric Stenosis," *Arch. of Ped.*, 1900.  
*Sprengberg*, L. H.: *Arch. f. Kinderheilk.*, Bd. xxvii.  
*Thomson*, John: "Congenital Hyper. of the Pylorus," *Edinburgh Hospital Rep.*, 1896. *Scott. Med. and Surg. Jour.*, 1897.  
*Treves*, F.: *Intestinal Obstruction*, New York, 1900.  
*Valle*, P. H.: *Situation du Cecum*, Thèse, 1900.



## CHAPTER VI.

### DISEASES OF THE RESPIRATORY TRACT.

#### GENERAL CONSIDERATIONS.

THE normal number of respirations in infants and children is as follows :

Immediately after birth . . . . .	44 per minute.
From the first to the sixth month . . . . .	24 to 36 " "
From the second to the fifth year . . . . .	20 to 32 " "
From the sixth to the tenth year . . . . .	20 to 28 " "

The ribs of some infants are very apparent to the eye, while in others they are so covered by a panniculus of fat as not to be seen. The normal chest has not the shape which it assumes later in life—that of a truncated cone. The lateral portions are quite straight and parallel. The chest is not flattened anteroposteriorly to the same extent as in the adult. In the newly born infant the transverse diameter is twice the length of the anteroposterior, while in the adult it is three times its length. In infants the superior border of the manubrium sterni is on a level with the mid-section of the first dorsal vertebra. In the adult it is lower by a body and a half of a vertebra.

The tendon of the diaphragm is horizontal in the newly born infant, and is on a level with the disk between the eighth and the ninth dorsal vertebra.

A rachitic chest may be conical anteriorly at the sternum (chicken breast). Some rachitic chests show a marked flaring of the lower ribs, with an incurvation above at the sides; they are flattened at the sides and taper toward the sternum. The sternum is the top of the truncated cone. Infants and children who have had several attacks of bronchitis, and who have some emphysema, show a marked fulness at the upper part of the chest beneath the clavicles.

*Skoliosis of the spine* may deform the chest, giving undue prominence to one side. Retraction occurs after the absorption of pleuritic effusions.

**Movements of the Chest.**—The movements of the chest may normally be irregular in rhythm; the sides move symmetrically.

In disease, especially in conditions of pressure on one side of the neck, one side of the chest may remain immobile, the other being retracted with each respiration to an exaggerated degree. I have

observed this condition after operations for retropharyngeal abscess in the neck, in cases in which the nerves in this region were pressed upon or injured, thus interfering with the normal action of the diaphragm.

In effusion into one side of the chest, there is diminished motion on the diseased side. Emphysema may restrict the normal movements.

Cheyne-Stokes respiration is seen in cerebral disease. After a deep and full inspiration the respirations become increasingly shallow until they are scarcely perceptible. A deep inspiration is then taken, and the respirations become more and more marked in the ascending scale, finally reaching the original force and depth. The cycle is then repeated.

In forms of pleurisy with effusion the intercostal spaces are retracted more than is normal at each descent of the diaphragm. This may be due to adhesions. The præcordial region may be drawn inward with the recoil of the heart, as is sometimes seen in adherent pericardium.

**Fremitus.**—The method of obtaining fremitus in children is described on page 24. It may be mentioned here that fremitus is well marked normally in the posterior axillary line and in the interscapular region.

**The Normal Limits of the Lungs.**—In the mammillary line on the right side to the sixth rib; in the mid-axillary line to the ninth rib. Posteriorly on the right side to the tenth rib; on the left side to the eleventh rib. Thus the limits are practically the same as in the adult subject (Symington).

The amount of lung-tissue above the clavicle cannot be mapped out in infants and children.

**Resiliency of the Chest-wall.**—The chest-wall in infants and children has a normal resiliency to percussion. The wall gives beneath the finger. This is a definite feature. In any disease of the chest which interposes fluid between the chest-wall and the lung this resiliency of the wall is absent. In infants and children, as in adults, there are normally :

- Pulmonary resonance ;
- Dulness varying to flatness ;
- Tympanitic resonance.

**Pulmonary resonance** is lower in pitch than in the adult. Anteriorly over the right infraclavicular region it is less marked than on the left side; the note is also slightly higher and of shorter duration.

**Dulness** is found normally over the heart, liver, and spleen; also, *anteriorly* on the right side from the fourth to the sixth rib. From the sixth rib to the borders of the ribs the note is flat. In the mid-axillary line on the right side there is dulness from the fifth to the seventh rib; from this point to the free border, the note is quite flat.



On the left side at the level of the sixth rib, just above the spleen, there is a narrow strip of relative dulness, due to the presence beneath the diaphragm of the left lobe of the liver (Fleischman) (see Spleen).

FIG. 95.



Strip of relative dulness described by Fleischman, and found just above the spleen, supposed to be due to the presence of the left lobe of the liver. Child, two years of age.

*Posteriorly* the supraspinous regions give dulness, but not so markedly as in the adult. On the right side, from the level of the seventh dorsal vertebra, extending downward, there is dulness due to the liver.

**Tympanitic resonance** due to the stomach is found normally in the left axillary line. It may in some cases extend high up in the axilla.

**Auscultation.**—As a rule, there is little difficulty in obtaining

the respiratory murmur and voice-sounds in infants and children—certainly not in the latter. The crying of unruly infants is useful in that it gives the fremitus and the quality of the voice-sounds. In some cases the infants are very quiet during examination, and unless they are teased into crying, definite information on these points cannot be obtained. The infant is caused to cry by gently squeezing the cheeks with the thumb and index finger.

**The Breathing.**—The respiratory sounds in infants and children are of an intensified vesicular quality; this so-called puerile breathing is normal and constant in children under twelve years of age. The quality of the vesicular murmur is probably caused by the better conducting qualities of the chest at this age. The elasticity of the lungs, which causes greater resistance to the inspiratory dilatation, is also a factor in producing the puerile quality of the respiratory sounds (Gutman).

**Types of Puerile Breathing.**—Puerile breathing in infants and children may be classified as follows:

*a.* The most common type is that in which the inspiration is coarse or intense in quality, while the expiration is vesicular and almost inaudible.

*b.* The second type of puerile breathing is that in which the inspiration and expiration are both of an intensified coarse quality.

*c.* The third type is that in which the inspiratory sound is low and vesicular, and the expiratory, coarse and puerile.

These types are found in infants and children at rest. If they are caused to cry, both the inspiratory and the expiratory murmur are of a coarse puerile quality. In some infants and children at rest, the inspiration and expiration are vesicular as in the adult. Puerile breathing is frequently confounded with bronchial breathing. It is, however, never tubular in quality. Bronchial or tubular breathing is marked on expiration; puerile breathing is generally so on inspiration.

During auscultation the sides of the chest are always compared. On the right side, beneath the clavicle and over the spine of the scapula, the expiratory murmur is more intense than on the left side. This should be especially remembered in cases in which disease of the right apex is suspected. The quality of the breathing in these regions approaches the bronchovesicular.

*Posteriorly*, the respiratory murmur may be heard as far down as the level of the eleventh dorsal vertebra. In some children the sounds are not so intense toward the base of the lung behind as higher up in the chest.

**Bronchovesicular breathing** is heard normally in the interscapular region in children as in adults. It has the same qualities as in the adult.

**Bronchial breathing** is heard normally over the trachea and upper



part of the sternum. It is also called tubular, tracheal, and over the larynx, laryngeal breathing.

**Forms of Dyspnœa.**—Though mainly of two types, pulmonary and laryngeal, dyspnœa may be caused by pain, fever, cardiac disease, and abdominal tumors.

**Pulmonary Dyspnœa.**—There is not only an increase in the number of respiratory movements, but also a change in the depth of each respiratory effort. In the dyspnœa of pulmonary disease, the region at the border of the ribs adjacent to the abdominal walls (peripneumonic groove) is drawn forcibly inward at each inspiration. In emphysema with asthmatic attacks, it will be noticed that during the attack the upper part of the thorax is immobile, the inferior part being drawn inward with each inspiratory effort. The presence of fluid in one side of the chest may be suspected if the side remains immobile, or if the intercostal spaces are drawn inward with each forced inspiration. A splenic or nephritic tumor may also, by simple upward pressure, immobilize one side of the chest.

**Laryngeal dyspnœa** will occur in any obstructive disease of the larynx. In addition to the phenomena of the pulmonary form of dyspnœa, there is a distinct retraction of the tissues at the situation of the suprasternal notch. There may also be laryngeal or croupy breathing.

While this is true in the majority of cases, I have also seen the retraction of the suprasternal notch, described above, present in the later stages of severe forms of acute pulmonary disease, especially in children; also in cases of emphysema in the asthmatic attack.

**Pain** will cause an increase in the number of respiratory movements. Thus the pain of an incipient pleurisy will cause an increased number of respirations which are more shallow than is normal. Peritonitic pain will also cause the respirations to become shallower and to increase in number.

**Fever** will, especially in infants and children, increase the number of respiratory movements to 40 or more, without the presence of any lung disease.

**Cardiac dyspnœa** is seen in those diseases of the heart which cause a retardation of the pulmonic circulation. The aëration of the blood in the capillaries of the lung is considerably interfered with under these conditions. Mitral disease, stenosis, and regurgitation cause dyspnœa not only for the reason given above, but also, in the later stages, on account of the bronchitis which is the result of the cardiac disease. Anæmia of cardiac disease is also accompanied by a slight dyspnœa, which is especially marked in children. The slightest exertion will sometimes cause angina and dyspnœa in children suffering from a slight cardiac lesion.

**Ascites and abdominal tumors**, or enlarged organs, such as the

liver or spleen, will cause dyspnoea, especially when patients are in the recumbent position.

In weak infants a few days old, who are the subjects of atelectasis and pneumonia, the upper part of the chest-wall moves very little, while the inferior portion of the chest and the upper part of the abdomen (peripneumonic groove) are drawn inward at each inspiration.

### ACUTE SIMPLE BRONCHITIS.

Bronchitis, acute and simple, is an affection of the larger and medium-sized bronchi. In very young infants the disease is apt to be very severe and to attack the smallest bronchioles; it is then called capillary bronchitis. A capillary bronchitis is really a bronchitis in which there is a certain amount of peribronchitic pneumonia. Acute bronchitis may occur at any period of infancy or childhood. It is, however, less common before the sixth month of infancy than during the period up to the third year, when its frequency diminishes.

**Causation.**—Bronchitis may be caused by an exposure to cold or wet or by traumatism to the mucous membrane of the air-passages through the inhalation of dust or irritating vapors. It occurs in the acute infectious diseases, such as malaria, scarlet fever, measles, r  theln, varicella, typhus and typhoid fevers, and frequently complicates pneumonia of the lobular or lobar type. Rachitis and syphilis predispose to attacks of bronchitis. The bronchitis of heart disease or nephritis should be regarded as of a different class.

**Pathology.**—The bronchi may be filled with a mucous, serous, purulent, or mucopurulent secretion, which is secreted by the epithelium of the mucous membrane and the mucous glands in the wall of the bronchi. In recent acute bronchitis the mucus is quite abundant. In the exudate on the mucous membrane of the bronchi and in the lumen, epithelial cells, leucocytes, and sometimes red blood-cells are found. The structure of the mucous membrane is infiltrated with small round cells to a greater or less degree. In some places the epithelial lining of the bronchi may be raised by exudate; in others there may be loss of the superficial epithelium. If the bronchitis lasts any length of time, there may be atrophy of the structures of the mucous membrane. In the severer forms of bronchitis which affect the smaller bronchi the peribronchitic connective tissue is infiltrated with small round cells. In these cases there is an inflammatory exudate in the surrounding alveoli of the lung. There is then peribronchitis or bronchopneumonia.

**Symptoms.**—In some cases the infant or child suffering from acute bronchitis will have a simple angina as an initial symptom. There is mild redness of the fauces with a slight rise of temperature



which may last a day or more. The cough which was present at first persists, and there may be slight disturbance of the bowels, the movements being green and containing large curds of undigested matter.

The cough may in aggravated cases give rise to occasional attacks of vomiting, especially immediately after nursing; at other times the coughing spells may cause the patient to cry. There is evidently pain, especially in the cases of bronchitis affecting the larger bronchi. The infant sometimes suffers from great difficulty in expelling the accumulated secretion. The attacks of coughing closely resemble those seen in old people who suffer from bronchitis. In many cases the infant or child is quite comfortable in the intervals between the coughing spells. In others the respirations are increased, and there may for some days be a slight evening rise of temperature, the patient showing signs of being seriously ill. In very young infants who are rachitic there may be a distinct drawing in of the sides of the chest and of the peripneumonic groove at each respiration. In cases of severe involvement of the smaller bronchi, there may be slight cyanosis of the lips and pallor of the surface.

In the severer forms of bronchitis, especially of the grippal variety, there is a distinct rise of temperature for several days. It may rise to  $102^{\circ}$ – $103^{\circ}$  F. ( $38.8^{\circ}$ – $39.4^{\circ}$  C.), or even higher, with a corresponding increase in the number of respirations and the pulse-rate. In weak and very young infants there may be little or no cough. The infant lies in a soporose state; does not nurse well or refuses the breast. Older children may run about and play while suffering from bronchial trouble; severe bronchial disturbance may appear to have little effect on the general health. Expectoration is very exceptional; a frothy mucus collects about the lips of young infants after an attack of coughing. In older children it may be very difficult to collect sputum, even if they are old enough to understand the necessity of expectorating the secretion. The conclusion has been that children swallow the expectoration; it is more rational to suppose that the efforts at coughing are not equal to raising any considerable quantity of secretion or that the amount of secretion in bronchitis is not so great as has been generally supposed. In many cases the cough is severer at night than during the day, but children cough and fall asleep immediately afterward, and therefore do not lose much rest. I have never met with a simple acute bronchitis ushered in by a chill or convulsion. I have, however, seen severe forms of bronchitis cause petechial extravasations on the skin, similar to those seen in pertussis. The petechiæ are apt to occur about the forehead and eyes of very weak infants.

**Physical Signs.**—In mild cases the respirations may be slightly above the normal; in severer cases there are signs of dyspnoea and

the respirations are increased in number. In very severe forms the peripneumonic groove may be drawn inward with each respiratory act. In capillary bronchitis the lips may show some cyanosis, the surface may be pale, and the finger-tips slightly cyanosed.

**Palpation.**—If the palms of the hands are placed in front and behind the chest, the so-called rhonchal fremitus may be elicited. The vibrations caused by accumulated secretion in the large and small bronchi give a sensation resembling that felt in stroking a purring cat.

**Percussion.**—In simple acute bronchitis, percussion may elicit nothing abnormal. If infants have suffered from repeated attacks of bronchitis, the note may, owing to a slight emphysema, be hyper-resonant or vesiculotympanic. In severe forms of capillary bronchitis there may be areas of peribronchitic pneumonia or bronchopneumonia, over which careful percussion will detect slight dulness with a resonant note.

**Auscultation.**—In a vast number of cases, bronchitis at the outset, gives on auscultation nothing but a rude respiratory murmur which is more markedly puerile than is normal. As the secretion accumulates there will be sonorous, sibilant, and subcrepitant râles, and also sonorous breathing. In the form called capillary bronchitis, with the subcrepitant râles there will be râles of much finer quality, resembling crepitant râles. The latter, which are unmistakable, are heard on inspiration, and appear to indicate areas of peribronchitic pneumonia. In newly born and weakly infants there are, in this form of bronchitis, areas in which the air is not heard to enter the lungs (atelectasis).

The **treatment** of simple acute bronchitis should be supporting and expectant. If the cough is harassing, a mild opiate mixture in combination with a small quantity of ipecac may be given. The following prescription has been found useful:

R	Tinct. opii camph.	. . . . .	℥j (4.0).
	Syr. ipecacuanhæ	. . . . .	℥℥ xxxij (2.0).
	Syr. tolutani	. . . . .	℥ij (60.0).
Sig.	Teaspoonful every three hours.		

The patients are allowed to be in the open air in fine weather, and the room should be well ventilated at night. In cases in which there is great relaxation of the mucous membranes, a dose of strychninæ sulph., grain  $\frac{1}{2000}$  (0.0003), may be given three or four times daily. The child is kept warmly clad, and wool is worn next the skin. Douching with cold water is to be avoided in acute cases. The oil-silk jacket may be worn, but it has no special superiority to warm clothing. Applications of oil to the chest are of no value. The drugs of the coal-tar series (antipyrin or phenacetin) should not be used, except that one dose may be given at the very outset to



relieve restlessness or headache. The bowels should be relieved by means of calomel or a saline cathartic.

In the subacute stage, syrup of ferric iodide may be given as a tonic for the mucous membrane. In very rachitic infants and children, cod-liver oil is indicated.

The treatment of so-called capillary bronchitis approaches very closely that of bronchopneumonia. The heart should be supported. Digitalis in the form of tincture is the most useful remedy. Strychnine, caffeine, camphor, and musk in form of powder, all have here their legitimate sphere.

The temperature, as a rule, needs no treatment. With older children, if the secretion is very profuse, carbonate of guaiacol is exceedingly useful and gives much relief.

### FIBRINOUS OR PLASTIC BRONCHITIS.

This is a form of bronchitis in which membranous masses or fibrinous exudate are coughed up at intervals. These masses may have the exact shape of the bronchi, or may consist of shreds or bands of membrane.

**Etiology.**—Bronchitis of this form complicates diphtheria and pneumonia, and also occurs in the acute infectious diseases—measles, scarlet fever, tuberculosis, erysipelas, typhus and typhoid fevers. It is found in diseases of the heart and lungs, and may result from traumatism through the inhalation of poisonous gases. The above are the secondary forms; the primary form of fibrinous bronchitis is obscure in its etiology, and is rare in infancy and childhood.

**Morbid Anatomy.**—The casts which are coughed up are cylindrical in shape and branched in the form of the larger and smaller bronchi. The larger ones may be hollow and cylindrical, while the smaller ramifications may be solid or thready. In other cases the whole cast is solid; small air-bubbles may be confined in the fibrinous cylinders. The casts may be 10–12 cm. in length, the extremities being nodular, thready, or flat. Under the microscope the casts are seen to be formed in layers; in the centre of the oldest layers are found epithelium of the bronchi, leucocytes, and bacteria. Spirals formed of fibrin are occasionally found in the expectorated masses, especially in the diphtheritic, pneumonic, and the so-called idiopathic cases.

**Symptoms.**—**Attacks of Dyspnoea.**—This form of bronchitis is characterized by attacks of dyspnoea and coughing. During the attacks clots of purulent fibrinous masses are expectorated, sometimes with a slight amount of blood. In spite of the expectoration of blood there are no signs of tuberculosis. The presence of blood

is probably caused by the detachment of the membranous casts from the walls of the bronchi. The expectorated masses may contain asthma crystals. In the intervals between the attacks, there may be symptoms of an ordinary bronchitis with mucopurulent expectoration, or there may be absolute freedom from symptoms.

The **cough**, which is present during the attacks, may be accompanied by a snarling or fluttering sound.

**Cyanosis** may be present during the attack to a marked degree or may be absent.

**Fever** is present in the acute form, but has no special characteristics.

**Splenic tumor** may be present.

The physical signs of bronchitis may be present with râles of all kinds. If the membranous masses hang detached in the bronchi, a snarling or flapping sound may be heard on auscultation.

The general condition of patients in the intervals and during the attacks varies greatly. In some cases it is fairly good.

**Complications.**—A tuberculous bronchitis or pneumonia may be a complicating condition.

The **diagnosis** is made from the presence of the fibrinous casts.

The **treatment** has thus far been very unsatisfactory; mercury, and also inhalations and sprays of all kinds have been tried by Biermer in the acute cases. Iodide of potassium is of value in the intervals. If diphtheria is present, the antitoxin is given.

### EMPHYSEMA AND CHRONIC BRONCHITIS OF THE LUNGS.

**Frequency.**—Emphysema is a condition frequently seen post-mortem in the lungs of infants and children (Steffen). No disease of the lungs runs its course without causing some emphysema. The condition is much more common in children than in adults, because it is favored by the peculiar structure of the lung during early life. Most of the forms of emphysema of the lungs of infants and children retrograde, allowing the lung to return to its normal state. Otherwise emphysema would be more common in adult life than it is. Clinically, emphysema combined with various forms of pulmonary disturbance, especially bronchitis, is very common in infants and children. My experience in this respect confirms that of Steffen and Osler. It seems to be common to certain classes of children, especially those of rachitic tendencies.

**Morbid Anatomy.**—Steffen has made a very careful study of the pathological condition in emphysema of the lungs of infants and children. The thorax has not the typical barrel shape seen in the adult, and occasionally found in older children. In younger



children, especially in those with rachitis, the sides of the lower portion of the thorax are incurved; the upper part of the thorax in front underneath the clavicles may be full and prominent. On opening the chest, the lungs are found to be inflated, to retain their form, and to show along the situation of the ribs a series of indentations due to pressure. The depressed portions may be denser than those raised, and show areas of circumscribed persistent pneumonia. In vesicular emphysema, air-vesicles may rupture into one another, giving rise to large sac-like formations which communicate with a bronchus. Some of the air-vesicles may rupture into the subpleural tissue. Vesicular emphysema rarely involves a whole lung or both lungs, but is localized to certain areas, such as the apices, anterior borders, or the lingula. The emphysematous areas are whitish, yellowish white, or reddish yellow, the color varying with the amount of blood contained. They are raised above the surface, are elastic and velvety to the touch, and crepitate with the air contained. In children, in contrast to the condition in the adult, the heart is rarely dilated, and the liver and kidneys rarely affected. This is due to the temporary nature of the process. Bronchitis, trachitis, and laryngitis may exist as primary or secondary conditions. It is not possible to consider emphysema in infants and children as an isolated condition. Since it is most frequently seen in pronounced bronchial affections, it will be convenient to consider it in connection with bronchitis.

**Symptoms.**—Some infants and children suffer from a chronic catarrhal bronchitis which is more or less present at all times, and which may be interrupted by attacks of acute bronchitis. Infants and children thus affected are more or less rachitic; some have lymphatism in the form of chronic hypertrophic rhinitis and also adenoids or enlarged tonsils. In the intervals between the attacks of acute bronchitis, the patients do not seem to suffer much constitutional disturbance. There is no fever, and no change in the respiration except that it assumes a noisy character. There is a cough which comes on at intervals, especially at night. The infants are pale, with rather flabby muscles, and may be fat, but impress the physician as being below the normal in point of strength.

**Physical Signs.**—If the bronchitis has persisted a long time, the upper part of the chest is, even in infants under the age of twelve months, abnormally full. The upper costosternal region is high and the intercostal spaces are filled out. In milder cases there are no signs to be detected on inspection.

**Palpation.**—There is distinct rhonchal fremitus felt anteriorly and posteriorly.

**Percussion.**—If there have been a number of acute attacks, there will be emphysema of a vesicular type, giving a hyper-resonant note. In pronounced rachitis the hyper-resonance is apt to be

marked. The area of relative cardiac dulness in older children is much diminished (Fig. 96).

**Auscultation.**—Voice-sounds are normal. The breathing is rude or sonorous. The respiratory murmur may be prolonged. There are sonorous, mucous, and subcrepitant râles.

A second set of cases of chronic bronchitis comprises those in which a condition of pronounced emphysema of a vesicular character is present, and in which there are distinct attacks of dyspnoea or asthma. These cases must be differentiated from the purely neurotic

FIG. 96.



Emphysema of the lung in a boy eight years of age; diminished cardiac area of relative dulness.

cases of spasmodic asthma. The latter condition is rare in children, and is not accompanied by chronic catarrhal bronchitis. The history of these cases is one of repeated attacks of acute bronchitis. The lung may in the interval be wholly free from signs of bronchitis. A condition of this kind is apt to be left in the lung after a severe attack of pertussis. The infants or children may bear the marks of rachitis, and are usually anæmic. In the intervals between the acute attacks of asthma, the general condition is good. There is no fever; there may be dyspnoea on exertion. An attack of asthma is precipitated by exposure to cold or wet. During the attacks infants



and children do not suffer much, although they show signs of marked dyspnœa. There are none of the typical signs of an attack of spasmodic asthma in the adult. An infant showing very marked dyspnœa will play in the arms of the mother. The lips may be cyanosed and the surface pale and cool. There is no temperature. There is in these subjects a tendency to develop a cough of a laryngeal type on the least exposure. Examination of the chest shows nothing except a prolonged rude respiratory murmur, while percussion will give a hyper-resonant note over the whole chest. Suddenly an attack of so-called asthma will develop, with all the physical signs given below. The onset of the attack is sometimes signalized by a slight rise of temperature,  $100^{\circ}$  to  $101^{\circ}$  F. ( $37.7^{\circ}$  to  $38.3^{\circ}$  C.), and an increase in the number of respirations, 32 to 36 per minute. On examination, the chest shows all the signs of an acute attack of bronchitic asthma. An attack lasts for from a few hours to a few days. The children usually play about and seem little disturbed by their condition.

**Physical Signs.**—*During an attack of spasmodic dyspnœa:*

**Inspection** shows a drawing inward of the supersternal structures on inspiration, and a depression of the peripneumonic groove. The upper part of the chest is high and filled out, and moves little on inspiration and expiration. The lower part of the thorax has also little movement. In rachitic children, there is not only drawing inward of the lower part of the thorax, but also a distinct incurvation of the lower ribs, caused by the repeated attacks of dyspnœa. The chest is moved as a whole. In children of seven or eight years the dyspnœa may be severe in the absence of cyanosis. These patients apparently suffer more than infants.

In older children, the chest has the typical barrel shape seen in the adult sufferer from asthma (Fig. 97). In one case, my notes describe a drawing inward of the intercostal spaces. Some cases have a constant cough and frothy expectoration.

**Palpation** gives rhonchal fremitus and faint cardiac impulse.

**Percussion** gives a vesiculotympanic or hyper-resonant note over the whole chest, and cardiac dullness obscured and diminished by the emphysematous lung.

**Auscultation** gives a prolonged expiratory murmur and sibilant and sonorous râles. Heart-sounds are feeble.

*Between the attacks of dyspnœa* the chest retains the above forms. There may be a slight constant dyspnœa or none at all. The patient feels quite well, and does not complain of the dyspnœa. The heart apex-impulse is diffused.

Palpation gives little or no rhonchal fremitus. Percussion shows a note hyper-resonant, but not as markedly so as during the paroxysm of dyspnœa. Cardiac relative dullness is obscured by the presence of emphysema.

**Auscultation.**—In older children the expiratory murmur may be prolonged or inaudible. There are signs of residual bronchitis, sibilant, sonorous, and subcrepitant râles, and in young infants, large mucous râles. The signs may be hardly noticeable or heard only in certain portions of the chest.

**Prognosis.**—In both forms of chronic bronchitis the prognosis *quoad vitam* is very good. The chances of ultimate restoration of the lung to the normal condition depend much on the mode of living and the power of the individual to outgrow the conditions of

FIG. 97.



Emphysema of lung; boy eight years of age; barrel-shaped thorax. Same patient as Fig. 96.

rachitis and lymphatism which exist in many of these cases. Many of these forms of chronic bronchitis disappear ultimately; the emphysematous form may persist into adult life.

The **treatment** of chronic bronchitis is directed toward improving the general tone of the economy and also the musculature of the heart. It must be assumed that in these cases the heart as well as the other organs suffers from a lack of power, to which may be attributed the relaxed condition of the circulation in the mucous membrane of the bronchi. Life in the open air, hydropathic treatment, and heart tonics, such as strychnine, will have beneficial effects. The mucous membranes are benefited by preparations of iron which contain iodine (syrup of the iodide of iron), freshly prepared and given



in large doses. Cod-liver oil is an excellent tonic in winter. The skin should be protected from extremes of heat and cold by suitable underwear. Moderate participation in sports in the open air improves the action of the heart. Running and gymnastics are to be preferred to bicycle-riding.

A dry climate will do much toward improving the condition of the lung. During the attack of dyspnea, iodide of potassium will be of service in alleviating the symptoms. This is the most useful remedy. It is also of great benefit when given in the intervals between the attacks. The other drugs used with adults are not indicated. I have seen good results follow the use of digitalis in the form of the tincture, in combination with the iodide of potassium. The heart is thus greatly aided in improving the circulatory conditions in the emphysematous lung. Rest from exertion is indicated during the attack, but patients may be kept out of doors if they will remain quiet. Codeine is most useful in allaying the cough. The administration of a large dose once or twice daily, is preferable to giving small doses at shorter intervals.

## BRONCHIECTASIS,

### Including Putrid Bronchitis.

Bronchiectasis, or dilatation of the bronchi, is not a very uncommon condition in infants and children. In most pulmonary disorders in these subjects, very slight dilatations of the bronchi may result. These have no clinical significance, and retrograde to the normal state in time. The marked dilatations are the congenital bronchiectasis and the acquired or inflammatory form.

**Congenital Bronchiectasis.**—This is a condition of the newly born infant which has been known to persist into adult life (Grawitz, Welch, Kessler, Fränkel). It generally affects one lung or a part of one lung. The lung structure is replaced by cystic formations which contain a serous fluid, in which are found nuclei and ciliated epithelium. The main bronchi may be cystic, with a system of minor cavities separated from the main cavity by a series of septa. In this way numerous recesses are formed. The walls of the cysts may be covered with several layers of cuboidal epithelium. No distinctive symptomatology has been reported in these cases.

**Morbid Anatomy.—Inflammatory Form.**—The inflammatory form of bronchiectasis may be sacculated, spindle-shaped, or cylindrical (vicarious). The cylindrical bronchiectasis shows the bronchus dilated into a cylindrical form. This dilatation may merge gradually or abruptly into the main bronchus. The spindle-shaped bronchiectasis is only a form of the cylindrical variety.

The sacculated bronchiectasis is the most common variety, and clinically the most important. It usually affects the smaller bronchi. A sac communicates with the trachea, and has no other outlet. The entry into the sac may be by way of a normal, a dilated, or a stenosed bronchus. If the infundibula are dilated, small cavities are formed (pulmonary vacuoles). In other cases the afferent bronchus may be obliterated, and the cystic formations are then of varying size. The wall of the bronchus leading to a cavity of this nature is in a state of catarrh, and may be thickened or infiltrated. The epithelium may be present only in spots. The infiltration may affect the walls of the alveolar septa. The mucous membrane may after a time become atrophic. The cartilages of the bronchi may also become atrophic and be replaced by connective tissue which may extend for varying distances into the lung substance, forming trabeculae. The epithelium of the bronchi may be replaced by pavement epithelium. The mucous membrane becomes thickened or is replaced by polypoid masses. The bloodvessels finally become dilated. There may thus be formed throughout the lung small aneurismal dilatations of the bloodvessels. The remaining lung tissue may be emphysematous or sclerosed as above. The pleura may be thickened.

**Etiology.**—Whatever the exact cause of a bronchiectasis, there is certainly a diminished resistance of the walls of the bronchus to the inroads of inflammatory processes. In order to explain the immediate formation of these cavities, Hoffman has assumed that a stenosis of the lumen of the bronchus (as shown by Fränkel and Lichtheim), must be produced by inflammatory processes and that under these conditions the repeated attacks of coughing produce dilatation. Such stenosis may have its origin in a peribronchitis or a pneumonia causing thickening of the wall of the bronchus. Pleurisy, chronic pneumonia, croupous or catarrhal, syphilis, and foreign bodies lodged in the lumen of the bronchi may be the direct cause of a bronchiectasis. Finally, there are the forms of bronchiectasis called primary, because their etiology has not as yet been explained.

**Symptomatology.**—The symptoms include expectoration, a cough, dyspnoea, deformity of the chest, and fever.

**Expectoration.**—There is expectoration of a mucopurulent character, which cannot be differentiated from the expectoration of some forms of bronchitis. In other cases, large quantities of a fetid, purulent material are expectorated. This expectoration may at times be mingled with streaks of blood, or there may be a distinct hemorrhage. In some cases there is a fatal hæmoptysis. Sometimes the sputum is profuse, exceedingly fetid, fluid, and purulent, and will on standing separate into a serous and a purulent portion.

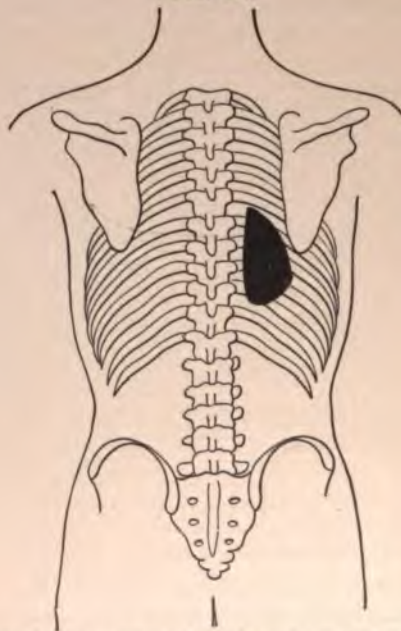
The cough may be occasional or, if the bronchiectasis exists in the apex of the lung, incessant. It is apt to be more marked in the





a localized empyema. Such cases show localized dulness or flatness, bronchophony, and absence of fremitus in a certain portion of the chest, generally at the lower portion behind. A needle, on being introduced, withdraws pus, which in the cases I have seen was mingled with air bubbles. On operation, the pleura is found to be normal. In 3 instances I found this to be true. The evidence of a bronchiectatic cavity lay in the persistence of signs and symptoms after the healing of the chest wounds. In all 3 cases the expectoration persisted in profuse quantities after operation (Fig. 99).

FIG. 99.



Showing bronchiectatic cavity in case of a girl eight years of age, with signs as noted in text.

The physical signs in all of my cases included a localized area of dulness or flatness, over which there was bronchophony and bronchial breathing, in some cases with gurgles. Above this area, over the base behind, there was on percussion a tympanitic note, indicating the enlarged bronchus containing air. Tuberculosis is excluded by the absence of tubercle bacilli in the sputum, but bronchiectasis and tuberculosis may coexist. In most of my cases there was a history of an antecedent attack of pneumonia. Exclusion of abscess of the lung is very difficult in severe cases in which the quantity of sputum is excessive. The bronchiectatic cavity in these cases is very large. With the bronchiectasis, there may be diffuse bronchitis and emphysema of the lung.



**Course.**—Some of the cases in which the bronchiectasis is not marked or progressive result in spontaneous recovery. In others there may be tuberculosis, gangrene of the lung, or empyema, as complications. A fatal hæmoptysis may close the scene of this very offensive affection.

**Treatment** does not give very satisfactory results. It includes the inhalation of balsams of all kinds, out-of-door life in high altitudes, and surgical interference including exposure of the lung and incision of the bronchiectatic cavity. The latter is a desperate remedy; in some cases it has resulted in fatal hemorrhage and in others has not afforded relief. A cure has resulted in a few rare cases in which there was a simple cavity in the lung near the pleural surface. The injection of these cavities with drugs has also been very unsatisfactory.

### LOBAR PNEUMONIA.

Lobar pneumonia or fibrinous pneumonia is an acute infectious disease, caused in the majority of cases by the *Diplococcus pneumoniae* (Fränkel). A few cases are caused by the *Bacillus pneumoniae* (Friedländer); others, by the *Streptococcus* or *Staphylococcus pyogenes*.

**Occurrence.**—Lobar pneumonia occurs as a primary disease or may complicate typhus fever, typhoid fever, influenza, rheumatism, malarial fever, erysipelas, osteomyelitis, meningitis, and nephritis. According to Keller, from 58 to 62 per cent. of all lobar pneumonias occur among children, the frequency among boys being greater (55.9 per cent.). Fully two-thirds of the cases occur during the winter and early spring. Pneumonia of any variety, and especially of this form, may occur in groups of persons or in small local epidemics. Without doubt, certain houses and rooms harbor the pneumonia poison for some time, as is evinced by the repeated occurrence of cases in certain places (Jürgensen). Cold favors the development of pneumonia by reducing the resistance of the economy to the invasion of bacteria, but it cannot be regarded as a cause of the disease.

**Age.**—Lobar pneumonia may occur at any age of infancy or childhood. Von Jaksch has shown that it occurs among young infants. My own experience confirms this statement. Out of 839 of my cases of pneumonia of all types, 582, or 69 per cent., occurred before the end of the second year; the greatest frequency was between the first and second years (282 cases). From birth to the sixth month the frequency is less than from the sixth month to the end of the second year.

**Sex.**—The male sex shows the greater number of cases (436

males, 403 females). Of 147 cases of carefully observed lobar pneumonia, 89 were males and 58 females.

**Seat of the Disease.**—Jürgensen shows that in 162 cases, both lungs were affected in 7.4 per cent. The right lung only was affected in 43.2 per cent. of the cases. When the right lung was attacked, the lower lobe was generally the seat of the disease (25.3 per cent.). The lower lobe of the left lung was consolidated in 35 per cent. of the cases.

Of 147 of my cases of lobar pneumonia, the right lung was involved in 79 cases and the left in 68; the upper right lobe was involved in 50 cases; the upper left, in 28. The upper lobe of either lung was involved in 78 cases, as against 61 cases of the lower lobes. The middle right lobe was involved in only 8 cases.

	Upper lobe.	Middle lobe.	Lower lobe.
Right lung . . . . .	50	8	21
Left " . . . . .	28		40

Pneumonia of the upper lobe is more frequent in children than in adults. According to Jürgensen, the greater frequency of pneumonia in the right lung may be attributed to the larger size of the right bronchus and the more direct communication with the lung.

**Morbid Anatomy.**—Lobar pneumonia in infancy and childhood is, as in adult life, distinguished by the occurrence of a fibrinous exudate in the alveoli of the lungs, bronchioles, and lymph-spaces. This exudate is composed of desquamated epithelium, leucocytes, red-blood cells, and fibrin. The proportion of leucocytes, red blood-cells, and fibrin varies greatly at different stages of the affection. A fluid exudate may be present if the quantity of fibrin is small. In such cases there is a lobar catarrhal process or an inflammatory oedema of the lung. The exudate begins with congestive hyperæmia. The lung is dark red and of increased consistency. With the appearance of coagulation there is produced a condition of hepatization in which the lung is solid, and has the appearance of liver. The bloodvessels are filled with red cells. If the vessels are less engorged, the lung has a grayish tint. This later stage, called gray hepatization, is the condition most frequently seen at autopsy. The hepatized lung does not contain any air, and on section shows a granular surface, the granules being the so-called pneumonic granules of the later stage of the disease. The pleura is as a rule inflamed. It is without lustre and may be thickened and covered with fibrin. There may be considerable serous or sero-purulent exudate in the pleural cavity. The extent of hepatization varies. It may involve a whole lobe, part of the lobe of a lung, or parts of both lungs. On inspection of the surface of a section, small yellow areas may be seen in the hepatized portions. These are areas poor in fibrin, and correspond to the situation of the bronchioles of the lung.



The bronchial nodes may be red and swollen, the bronchi being the seat of inflammation. The bronchioles may be filled with fibrin and red blood-cells.

Resolution occurs on from the seventh to the tenth day of the disease. At this time liquefaction of the inflammatory products which are eliminated by expectoration occurs. Complete restoration of the lung to the normal may occur between the second and the fourth week, at which time the periphery of the alveoli may be found to be rich in cells. There may still exist catarrhal processes which have succeeded the fibrinous changes. The pleura may remain thickened and be the seat of adhesions.

An unfavorable or malignant ending, such as gangrene or supuration, is rare, and is as a rule due to some mixed infection favored by an old bronchiectasis or putrid bronchitis. Unless a tuberculous infection occurs, caseation in lobar pneumonia is unknown. Induration of the lung, cirrhosis or carnification, is a peculiar condition which may occur from the fourth to the tenth week. The lung assumes a beefy red appearance and is tough, hyperæmic, and infiltrated with small round cells. The alveoli enclose a large number of connective-tissue cells. There is a proliferation of newly formed bloodvessels in the septa of the lung. The bronchial, peribronchial, and pleural tissues are proliferated. Induration of the lung by pleural adhesions results. The alveoli of the lung may be replaced by connective tissue and epithelium. Induration may take the form of bands of connective tissue, which may extend from the pleura into the lung, enclosing areas of lung-tissue.

**Bacteriology and Etiology.**—The pneumococcus of Fränkel is now recognized as the etiological factor in lobar pneumonia. As has been mentioned, the *Bacillus pneumoniae* of Friedländer is found in a small number of cases, with the pneumococcus or with other bacteria. The *Streptococci pyogenes* and the *Staphylococcus pyogenes* are sometimes found, as well as the *Bacillus typhosus*. In the cases of secondary infection, the *Diplococcus pneumoniae* or the *Staphylococcus pyogenes* is found. In the majority of fatal cases, Kohn found the pneumococcus circulating in the blood. The cases which show the diplococcus in the blood and which recover, do so with complications. In a recurrent pneumonia of infancy, Perutz found an osteomyelitis of the joint, caused by pneumococci. In one of my cases which was followed by bilateral empyema, there was a peri-articular abscess containing pneumococci. According to Landouzy and Netter, the pneumococcus is capable of producing suppuration without the intervention of streptococci or staphylococci. Cases of severe icterus are due to the hæmolytic action of the pneumococci on the blood. Gaillard has shown that the enteritis in pneumonia is caused by pneumococci.

**Symptomatology.**—There are forms of fibrinous or lobar

pneumonia which present the same symptomatology in children as in the adult. On the other hand, certain sets of symptoms referable to the nervous system and intestinal tract, as well as the character of the variations in temperature, are peculiar to infancy and childhood.

The disease may be ushered in by a chill, which may be severe or only amount to a sensation of chilliness. Susceptible subjects may, with the rise of temperature, be attacked with convulsions. Other patients pass into a stage of delirium lasting for days. Cases of pneumonia ushered in with cerebral symptoms are apt to mislead the physician, especially if meningitis has been recently prevalent. There are also cases, especially in children, in which there has been a preceding bronchitis. These should not be regarded as being of necessity cases of bronchopneumonia. Sometimes the chill is coincident with a sharp attack of enteritis. The character of the invasion will thus vary with the severity of the infection and the susceptibility of the subject. After the initial chill, there is in the simple cases a sharp rise of temperature. The height of the fever varies, and in young infants is apt to mount to  $106^{\circ}$  F. ( $41.1^{\circ}$  C.). There are cough and considerable dyspnea, varying with the extent of lung involvement. In infants and children the dyspnea is quite apparent to the eye of the observer, and will prompt him to surmise that the lung may be involved. Older children have a distressed expression.

The patient complains of pain, which is in many cases referred to the side affected. In younger children the pain is quite frequently referred to the epigastrium, but sometimes to the region of the abdomen low down, or to the right side of the abdomen low down over the situation of the vermiform appendix. Pain is apt to be referred to this region in cases of lobar consolidation of the lower portion of the right lung. These are often, in the early stages, diagnosed as cases of appendicitis. The face is pale or quite flushed. The dyspnea may be slight, but is quite marked in some severe cases. Even if both lungs are involved, it may not be intense. There is a cough. In older children there is expectoration of rusty sputa. Infants and young children swallow the sputum. Infants cry with each paroxysm of coughing; older children complain of pain. Sometimes infants and children vomit with each attack of coughing. After the fever has persisted with these symptoms for from five to nine days, there comes in the vast majority of cases a fall of the temperature—the so-called crisis—which may take place within from three to six hours, or may extend over thirty-six hours. The fall of temperature may be followed by a temporary rise of a few degrees (Fig. 186)—the so-called post-crisis: within a few hours it then falls to the subnormal, where it remains for a few days after the crisis, finally



rising to the normal and remaining at that point throughout convalescence. The temperature may fall by lysis, that is to say, by reaching with gradual remissions the normal, or as a rule the sub-normal, within from forty-eight to seventy-two hours.

**Consideration of Individual Symptoms.**—**THE TEMPERATURE.**—The temperature-curve in lobar or fibrinous pneumonia may be of several distinct types. In the majority of cases the temperature remains persistently high for the whole period of the illness. There are morning remissions of a degree or more, but the afternoon or

FIG. 100.

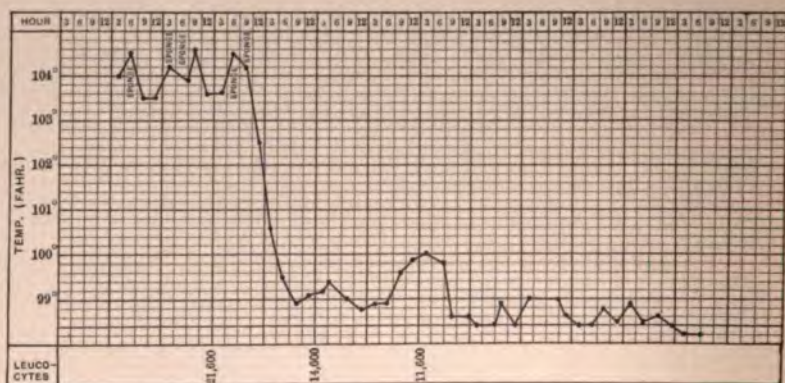


Lobar pneumonia; pseudocrisis and crisis. Leucocyte count before and after crisis indicated. Boy four years of age.

evening rise may reach 104°, 105°, 106° F. (40°, 40.5°, 41.1° C.). In a typical case the morning remissions are not so great as those in pneumonia of the bronchopneumonic type. The crisis is not as a rule preceded by a rise. The drop of the temperature at the crisis in a fairly typical case may begin at 9 A.M., and the temperature may be subnormal at 9 P.M. of the same day (Fig. 101). In another form, crisis may be rapidly followed by a temporary rise in the temperature, not due to any reinfection of the lung, but to a slight post-pneumonic toxæmia. The temperature will in such cases reach the subnormal within thirty-six hours.

Another very distinct form of temperature-curve is the remittent. This temperature-curve is at first glance exactly similar to that of bronchopneumonia. The remissions in the morning may reach the normal within a fraction of a degree. Such cases may also show at the terminal end of the curve a critical drop to the normal. In other cases the fall of temperature at the beginning of convalescence takes place by what is known as lysis (Fig. 102). In other words, the temperature reaches the normal or subnormal by remissions of temperature in a gradually descending scale extending over two or more days. Some cases show a remission of the temperature which begins at the ninth day of the disease, and is not completed until the fifteenth day. This is occasionally seen in cases in which there are

FIG. 101.



Lobar pneumonia, right lung, lower lobe. Crisis on the eighth day. Leucocyte count indicated. Female child, two years and five months of age.

apparently no complications. The more common type is that in which the lysis begins on the seventh or eighth day, and is completed in two or three days. Of 57 cases of lobar pneumonia in which a reliable history could be obtained, the temperature fell by crisis in 36 and by lysis in 21 cases. The crisis, as a rule, occurs from the fifth to the ninth day of the disease (60 per cent. of my cases). After the lysis or crisis there may be a slight daily rise in temperature of a degree or even less, probably indicative of a very mild form of post-pneumonic pleurisy. The temperature in such cases falls gradually, and in four or five days reaches the normal (Fig. 103).

The subnormal temperature after the crisis or lysis is quite a common phenomenon. I have learned not to fear this symptom, but to regard it as favorable (Fig. 104). A subnormal temperature may persist for days, or even a week or longer, and not uncommonly, especially in fibrinous pneumonia which has run a sharp or moderately severe course, is accompanied by irregularity or abnormal



slowness of pulse. A slow pulse (bradycardia) which is at the same time irregular is apt to be alarming to the physician, but I

FIG. 102.



Lobar pneumonia, right lung, lower lobe; temperature falls by lysis. Leucocytosis indicated in the chart. Female child, four years of age.

FIG. 103.



Lobar pneumonia, right lung, middle lobe; effusion into the pleura. Temperature after crisis due to pleurisy. Boy, eight years of age.

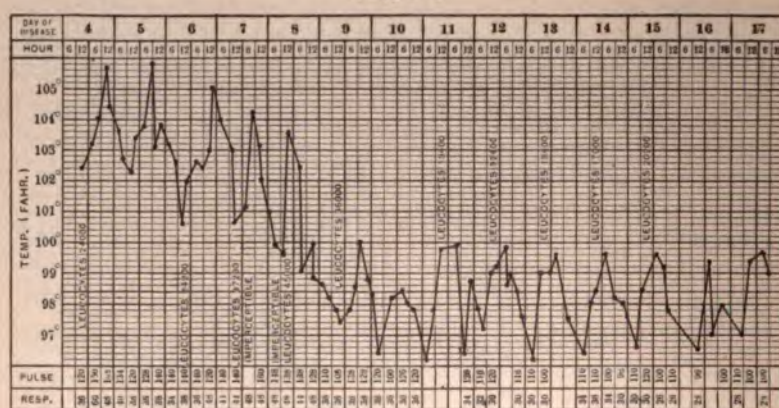
have never seen any ill effects in these cases if they were treated in a rational manner. Such conditions of pulse and temperature

should be regarded as a reaction from the toxæmia which has affected the heart muscles.

At the crisis in lobar pneumonia I have, in exceptional cases, seen the temperature drop within an hour from 103° to 94° F. (34.4° to 39.9° C.) and the pulse to 48; within an hour the temperature rose to 96° F. (35.5° C.) and the pulse to 70. The temperature gradually rose, so that within seven hours it was again 99° F. (37.2° C.) in the rectum, the pulse 96. The symptoms of mild collapse may accompany the pronounced fall.

**THE COUGH.**—Some infants and children cough very little; in others the cough is a very harassing symptom. There is no sputum even with the older children, or only after the crisis; pain

FIG. 14



Lobar pneumonia, right lung, upper lobe: remittent temperature-curve; prolonged subnormal temperature intermittent in character; recovery. Female child, two years and six months of age.

accompanies the cough, and may be suspected if the infant or child cries when it coughs. The pain is referred to the side of the chest, to the epigastrium, or to the region of the appendix. The pain referred to the appendix in cases of lobar pneumonia is probably radiated from a diaphragmatic pleurisy.

**DYSPNOEA.**—Infants and young children show marked dyspnoea. The alæ nasi are dilated and the peripneumonic groove is depressed with each inspiration. In very severe dyspnoea in young infants, there may be a drawing inward at the suprasternal notch. This occurs even in the absence of any laryngeal disturbance, and frequently simulates laryngeal stenosis.

**NERVOUS SYMPTOMS.**—The cerebral symptoms may at the outset simulate those of cerebrospinal meningitis. There are delirium, rigidity of the muscles of the neck, and even opisthotonos. There may be no true meningitis. Older children may have a low, mut-



tering delirium during the whole course of the disease. Near the crisis and just before the fall of temperature, I have in a few cases seen maniacal delirium, in which the patients were very noisy and attempted to get out of bed. I have seen cases of melancholia with crying spells during convalescence in female children, and also in boys. These symptoms all subsided in time and the patients were eventually fully restored.

**THE BLOOD.**—It has been noted by Tumas and von Jaksch that in pneumonia of the fibrinous variety there are a marked leucocytosis and an increase in the multinuclear leucocytes, which is especially marked at or near the crisis. The proportion of leucocytes to the red blood-cells in the cubic millimetre may reach 1:40 to 1:70. Ehrlich believes this leucocytosis to be a very constant occurrence in typical pneumonia. Billings has investigated the relationship of the leucocytosis to the prognosis more fully. His work will be referred to in the consideration of the prognosis. My own experience covers about ninety cases of fibrinous and bronchopneumonia, examined with reference to leucocytosis. Leucocytosis is present in both forms of pneumonia in infancy and childhood, but is more marked in the fibrinous forms, the number of leucocytes to the cubic millimetre being about twice as great as in the catarrhal forms. There was marked leucocytosis in the fatal cases of both forms of pneumonia. The increase of the leucocytes in the fibrinous forms was especially marked at the time of the crisis. In the bronchopneumonic forms the leucocytes were also high at or about the time of the drop in temperature. The diminution of the number of leucocytes was in both forms marked either just previous to or after the fall in the temperature. From the observations of Billings and Ewing, it must be concluded that leucocytosis is a favorable sign in fibrinous pneumonia. It does not, however, as Ewing believed, bear any exact ratio to the extent of lung involved. I have found a much higher percentage of leucocytes to the cubic millimetre in children than Ewing found in the adult. This is probably due to the fact that any leucocytosis is more marked in infants and children than in the adult subject. The absence of leucocytosis is certainly a grave prognostic sign, but the presence of marked leucocytosis in children does not in my experience preclude a fatal issue.

**Physical Signs.**—The signs obtained by physical examination of the chest in fibrinous pneumonia of infants and children resemble those of the same condition in the adult. In forms of bronchopneumonia or catarrhal pneumonia in which areas of considerable extent are consolidated the signs will closely resemble those obtained in the fibrinous form. The physical signs of lobar or fibrinous pneumonia are classified as those of the first, second, and third stages of the disease.

**First Stage, Stage of Engorgement of the Lung.**—On INSPECTION the signs of dyspnoea above noted are found.

PALPATION at this stage will in an uncomplicated case give no signs, even over the affected area. If bronchitis complicates the case, rhonchal fremitus may be obtained. At this stage the difference in fremitus between the affected and the unaffected side of the chest is not perceptible.

AUSCULTATION.—In the first stage of the disease auscultation may discover a rude respiratory murmur on the healthy and diseased sides which is more marked in the latter and on inspiration. The pathognomonic sign at this stage is the crepitant râle, which is sometimes easily found and is at others very elusive. It may be present before an attack of coughing, and disappear after the bronchi have been cleared, and is, as a rule, heard over a very limited area. It is therefore necessary to examine the chest very carefully in front, behind, and in the axillary line for this sign, before deciding positively as to its presence or absence. It may be present for a few hours only.

PERCUSSION will at this period give slight dulness over the affected area of lung. The dulness may be slightly tympanic. This is caused by the fact that at the outset of consolidation there is still some air in the affected area. Under these conditions there may be what is known as tympanic dulness. This condition is especially found in young infants, in whom the chest-wall is thin, and in whom sounds are very well obtained by gentle percussion.

**The Second Stage, Stage of Consolidation.**—If the lower portion of the right lung is affected, we shall get by palpation in front over the upper part of the chest nothing abnormal; over the lower part of the chest in front there will be an increase of the vocal fremitus, which is also apparent behind. Percussion over the upper part of the right lung will give a vesiculotympanic note in front and behind. The unaffected side will give normal pulmonary resonance. In exceptional cases the percussion-note over the upper lobe of the lung in front may give the so-called cracked-pot sound. In front, behind, and in the axillary line over the lower lobe which is affected there is dulness—not at first complete. When consolidation is complete, the dulness is quite marked. In cases in which some pleuritic effusion exists over the consolidated area behind, the percussion-note may be quite flat. In cases in which the upper lobe is consolidated there will be signs of consolidation, while lower down the note is exaggerated or vesiculotympanic over the unaffected mid-region of the lung, and over the base there will also be marked dulness. This lower area of dulness should not be regarded as a sign of consolidation. It is really due to the accumulation of a small amount of serous effusion in the lower part of the pleural cavity as a result of the complicating pleurisy.



AUSCULTATION will in this stage give bronchial voice and breathing over the affected area of the lung; over the unaffected lung the respiratory murmur, especially the inspiratory sound, is harsh. This harsh inspiratory sound is quite common in children, and is frequently mistaken for bronchial breathing. Bronchial breathing is tubular in quality on inspiration and expiration. In this stage, if the upper lobe of the lung is also involved and there is some pleuritic effusion in the chest, the respiratory murmur may be much weakened over the lower region of the chest behind.

The voice also has a tubular or bronchial quality over the consolidated area. The intensity of the voice may be diminished over the lower portion of the chest if pleuritic effusion is present with consolidation of the upper lobe. Pleuritic râles may in this stage be heard over the whole side of the chest.

**Third Stage.**—The third stage, that of resolution, is sometimes delayed, some days elapsing after the crisis before appearance of the sign pathognomonic of this stage—the so-called *râle redux*. This *râle* has the same qualities as that heard in adults at the same stage. In children it is sometimes present for only a short time, and is not heard over any considerable area of the lung. I have known the temperature to be subnormal for two days or more before its appearance. The other sign, which is less important, is a distinct diminution of the fremitus until it reaches the normal intensity over the affected area of lung. The percussion-note becomes less dull, assuming the vesiculotympanic quality. Repeated auscultation reveals, in addition to the *râle redux*, a gradual return of the voice and breathing to the normal, which sometimes takes weeks. The tubular quality of the voice and breathing over the affected area of lung may persist long into convalescence. It is probably not caused by any actual persistence of consolidation, but by a continued hyperæmia of the lung. The lung under these conditions is denser and conducts sounds from the bronchi with greater intensity than the healthy lung. If pleurisy has been present to any extent, there may, after the disappearance of the signs of consolidation, be signs of dry pleurisy or those of effusion.

**Pneumonia of an Unusually Short Course.**—Leube and Weil have recorded in the adult typical pneumonia of the fibrinous variety and of very short duration. Some of these cases exhibit the chill, fever, pain, and crisis, with other signs of physical involvement of the lung, within twenty-four to thirty-six hours. Jürgensen has recorded short lethal pneumonias of the fibrinous variety in the adult. The cases of Levy and Jürgensen were fatal within twenty-four to thirty-six hours. I have never met such cases of fibrinous pneumonia in children. In cases running such a course there is doubt as to whether the signs obtained over the chest may not have been connected with a preceding attack. Henoch has, however,

met a few cases which ran a rapidly fatal course with the whole symptomatology of lobar pneumonia including physical signs, in forty-eight hours.

**Complications.**—Among the complications of fibrinous pneumonia in infants and children are otitis, pleurisy, pericarditis, endocarditis, empyema, and meningitis. Some writers record peritonitis; I have not met a case. Gastro-enteritis is quite a common complication.

**Otitis** is common, its frequency varies in different epidemics. It affects younger children and infants more frequently than older subjects. The temperature in these cases becomes more markedly remittent and remains higher for a greater length of time than in the uncomplicated cases. I have frequently suspected otitis from a study of the temperature-curve, which is not, however, an altogether reliable guide. Suppuration in the pleura will give a similar curve. Therefore, in a concrete case of persistent high temperature-curve with morning remissions, otitis should be suspected, but not positively diagnosed without careful exclusion of other complications. Otitis as such does not seem to give any striking symptoms of pain. The patient may without warning present perforation of the drum of one or both ears and a purulent discharge. The temperature will then fall to the normal. *Diplococcus pneumoniae* has been found by a number of observers in this discharge. The otitis is of a benign nature.

**Meningitis** occurs in a number of cases, and may usher in the disease. I have seen it persist for weeks. The prognosis in this form of meningitis, if it assumes the cerebrospinal type, is graver than when it occurs as a primary disease, with the intracellular *diplococcus* of Weichselbaum as a causative factor. Netter seems to have met a larger number of cases of the pneumococcus form of meningitis than any other author. The cases of meningitis complicating pneumonia should not be confused with those presenting cerebrospinal symptoms. The cerebrospinal symptoms seen at the outset or at the crisis in some cases of pneumonia do not last for any great length of time, and do not present the true symptoms of meningitis.

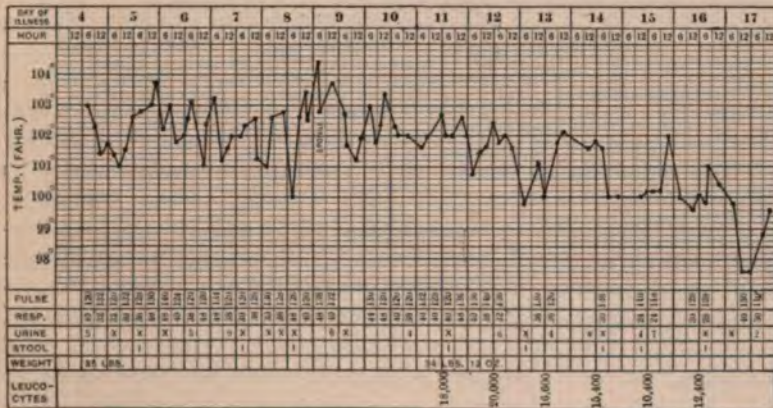
**Pleurisy and Empyema.**—Many cases of fibrinous pneumonia show a dry pleurisy sometimes persisting for a long time after convalescence. Of greater moment are the cases of pleurisy with effusion, which follow a lobar pneumonia. In these, there is always the danger that the exudate may eventuate in an empyema. The duration of the exudate is no guide in determining whether it is of a serous or a purulent nature. It is frequently found that after a pneumonia has run its course the temperature remains raised a degree or more toward evening. Such a rise in temperature may, in the absence of signs of fluid, indicate a dry plastic pleurisy (Fig. 105). On the other hand, if there are signs of fluid and the temperature-curve



shows irregularities of rise, empyema may be present. I have met empyema without any rise of temperature in infants who showed the physical signs of fluid in the chest. These points will be more fully discussed under the head of Empyema.

**Pericarditis.**—I have seen pericarditis in infants who died of a fibrinous pneumonia, but the diagnosis was not made during life. Von Jaksch notes such cases. In older children, pericarditis is a complication found in cases of fibrinous pneumonia which have simultaneously developed empyema. Such cases are very uncommon. In the form of pericarditis which I have seen in infants, the quantity of effusion has not been sufficiently great to enable a diagnosis to be made with certainty, and the râles in the lung obscured the friction-sounds in the pericardium if they were present. Purulent pericarditis in these subjects is very fatal

FIG. 105.



Lobar pneumonia, lower lobe, left lung; complicating pleurisy; temperature falling gradually to the normal. Leucocyte count indicated. Boy, five years of age.

under such conditions. In older children I have seen pneumonia combined with a fibrinous pericarditis pure and simple, without fatal issue.

The **prognosis** of lobar pneumonia varies within certain limits. Text-books give statistics taken from hospital cases, notably the most unfavorable material. Henoch gives the mortality of his cases at 5 per cent.; Baginsky, at 8 per cent.; Holt, at 12 per cent.; my own hospital cases during the past year showed a mortality of 8 per cent. On the other hand, in private practice death from an acute fibrinous pneumonia rarely occurs in a child previously healthy and living in good surroundings. The mortality is influenced by the season of the year, being greater from December to February, and by the presence of an epidemic. If pneumonia is prevalent during an epidemic of influenza, the mortality will increase. Pericarditis or

complicating empyema influence the death-rate. The previous condition of the patient, the mode of feeding (whether by the breast or the bottle), and a rachitic or marantic condition, affect the prognosis. The age of the patient is also an important factor. Infants under one year of age are in greater danger than older ones. The prognosis is best from the third to the tenth year. The younger the bottle-fed baby, the more serious the complication of empyema. In making a prognosis in any concrete case, the physician should be guided by the extent of lung involvement and the general condition of the circulation. If one lobe alone is involved and there is an absence of bronchitis in the unaffected lung, the outlook is good. If the heart action is good and there is an absence of cyanosis, recovery can be predicted even if the temperature be high. If, on the other hand, the lysis or crisis is delayed and the dulness or flatness involves a whole side of the chest, in the presence of signs of a weak heart the prognosis should be made with caution. Meningitis of pronounced type is grave. Pericarditis in young infants and children is a complication invariably fatal.

The **diagnosis** of lobar pneumonia in infancy and childhood ordinarily presents few difficulties, but is not easily made if in addition to the pneumonia there is an effusion in the chest. The diagnosis should never be made early in the disease without positive signs.

The crepitant râles sometimes escape observation. The physician should then wait for the appearance of dulness or bronchial voice and breathing before arriving at a conclusion as to the presence or absence of consolidation. Cases of influenza with a harassing cough are frequently diagnosed as central pneumonia. A pneumonia which is central will give physical signs. If after the time set for the crisis or lysis, the temperature persists and becomes remittent, careful examination should be made for evidences of fluid in the chest. The nature of the fluid should be determined by exploration with the aspirating needle, if the fever does not subside and if the dyspnoea increases. A chest effusion in infants and children is apt to be purulent.

The cerebral cases present difficulties of diagnosis. Convulsions, delirium and rigidity of the neck, accompanied by high fever and a cough, with increase of the pulse-rate and the number of respirations, indicate the necessity of making a very careful examination of the chest.

In cases which begin with a lobar pneumonia, typhoid fever may be suspected if, after the first days of illness, a roseola or an enlargement of the spleen develops with a continuance or gradual rise of temperature. In such cases the presence of an epidemic of typhoid fever and the Widal blood reaction will be of service in clearing up the diagnosis.



The **treatment** of lobar pneumonia is pre-eminently expectant. The disease is self limited, and complications cannot be prevented. The temperature should be treated within certain limits, and the heart and the strength of the patient supported. The temperature should be treated not with a view to its actual reduction, but in order to mitigate its ill effects. Infants and children will be less affected by a temperature of 103° F. (39.4° C.) lasting during the time while a pneumonia runs its course than by the same temperature in typhoid fever. The toxæmia of pneumonia is of a more benign character. Cold applications are relied on to reduce the temperature.

**Hydrotherapy.**—Sponging is efficient in cases in which the temperature does not generally range above 104° or 104.5° F. (40° C.). The younger the infant the less energetic need it be, for a temperature of 104.5° F. (40° C.) is not high for an infant under two years of age. I content myself with sponging of the body with water at 80° F. (26.6° C.), to which some alcohol has been added. If the temperature remits a degree or more during the twenty-four hours, there will be less need of sponging. The temperature should never be taken more often than every three hours. If it is above 103.5° F. (39.7° C.), the patient is sponged for fifteen minutes and then given absolute rest for three hours. Frequent sponging is pernicious. Some infants when sponged with water at 80° F. (26.6° C.) become blue, the pulse becoming rapid and thready. With these subjects a warm bath at a temperature of 105° to 107° F. (40.5° to 41.6° C.) is stimulating. It supports the strength and certainly lessens the ill effects of the temperature, although it may not reduce it palpably. I do not use the full cold bath in the treatment of lobar pneumonia in infants and children. If the temperature reaches 105°–106° F. (40.5°–41.1° C.), a full bath of the temperature of 85°–90° F. (29.4°–32.2° C.) or higher may be given, certainly never lower.

One of the most useful methods of hydrotherapy in the treatment of pneumonia in young infants is the so-called chest compress (page 35). These compresses renewed every hour will cause the restlessness to diminish, the heart action to improve, and the patient to fall into a quiet slumber. The actual reduction of temperature is not so marked as the favorable effect on the general condition of the patient. The application of compresses is discontinued if the temperature falls below 103° F. (39.4° C.).

**Medicinal Treatment.**—The heart action if good needs no attention. At most, a limited amount of wine or whiskey is administered. Infants may receive half a drachm (2.0) every few hours; older children, a drachm (4.0). Whiskey should not be given as a routine remedy. If the temperature is high necessitating hydrotherapy, and the pulse is above 120, whiskey should be given. If the pulse

is high, 150-160, a few minims of the tincture of digitalis may be given to older children. Younger children rarely need more than half a minim every two to three hours. If the pulse-rate is reduced after the administration of digitalis, the drug should be discontinued before the pulse drops below 100. There is no doubt that its effect is more cumulative in some subjects than in others.

Strychnine is of value in the treatment of pneumonia, not so much in the cases with rapid as in those with slow and irregular pulse. Infants will bear grain  $\frac{1}{200}$  to  $\frac{1}{150}$  (0.0003 to 0.0004) every three hours, for days.

Caffeine is of great value in the treatment of irregularities of the heart which indicate a myocarditic process. The pain is the result of a pleuritic process.

The local application of iodine or mustard paper is an efficient counter-irritant. If the cough is troublesome, codeine in moderate dosage is the most useful remedy.

I never make use of morphine with infants and children. In young infants the milder preparations of opium, such as camphorated tincture or the wine, are most useful. Four minims (0.25) of the camphorated tincture of opium every two or three hours will be found efficient in children under two years of age. To older children a small dose of codeia may be given several times daily if needed. The aim is to alleviate the pain and cough.

The bowels should be evacuated daily; for this purpose hydrarg. cum creta is one of the best remedies. Grain v (0.3) may be given. Infants should receive an enema daily.

If gastro-enteric disturbances are present, the giving of milk should be discontinued and the same procedure followed as in primary gastro-enteritis.

Tympanites is sometimes troublesome, especially in young children. The best remedy is a high enema twice daily of salt solution, to which one or two teaspoonfuls of peppermint-water have been added. The passage of a soft catheter is not effective, nor are the turpentine stupes of any value.

The delirium, sometimes amounting to an acute mania, which appears just before the crisis in some cases, is best controlled by rectal administration of bromide of potassium and chloral hydrate. I have sometimes been forced to keep the patient under the influence of these drugs for a few days. The post-pneumonic melancholia seen in children is best treated by the administration of strychnine and the enforcement of perfect quiet.

Should signs of extreme cardiac weakness set in with threatening œdema of the lung and paralysis of the right ventricle, nitroglycerin is of great value. Infants will bear grain  $\frac{1}{200}$  (0.0003) every three hours. If in these cases cyanosis is present, oxygen is administered, preferably that containing 20 per cent. of nitrous oxide. It is given



to infants, every half hour for five or ten minutes at a time by means of a cone.

**Hygiene.**—The patient should be isolated if possible. The room should be ventilated and its temperature kept at 68°–72° F. (20°–22.2° C.).

The sputum should be received in pieces of gauze, which are burned. The mouth and teeth should be cleansed twice daily with a piece of soft linen and a solution of boric acid. In the intervals between feedings the tongue is kept moist by frequent draughts of water.

### BRONCHOPNEUMONIA.

Bronchopneumonia is the prevalent type of pneumonia occurring before the fifth year, but there are also many cases of lobar fibrinous pneumonia during the periods of infancy and early childhood.

Bronchopneumonia occurs both as a primary and a secondary disease. As a primary disease it is most frequent during the first two years of life. Of 605 of my cases of bronchopneumonia, the incidence in regard to age was as follows :

	Cases.
One to three months . . . . .	32
Three to six months . . . . .	68
Six to twelve months . . . . .	207
One to two years . . . . .	298

These figures correspond within certain limits to those of other authors, although Holt places the greatest frequency between the sixth and the twelfth months.

**Sex.**—Of the 605 cases, 322 were males—a statement corresponding to that of Jürgensen in regard to lobar pneumonia.

**Season.**—The greatest frequency is during the winter months, when there are epidemics of influenza during which many primary and secondary cases of bronchopneumonia occur.

**Surroundings.**—The herding together of the poor certainly has a tendency to increase the prevalence of bronchopneumonia among them. If we believe in the epidemiological aspects of pneumonia, it is easy to account for the greater frequency of the disease among the poor: the greater number of their children are rachitic, syphilitic, marantic, and ill-fed, and thus have increased susceptibility to infection.

Secondary bronchopneumonia occurs as a complication in the exanthemata (measles, scarlet fever, typhoid fever), diphtheria, pertussis, and influenza. By far the greater number of cases occur as a sequence of ordinary bronchitis.

**Etiology and Bacteriology.**—Weichselbaum first demonstrated that the pneumococcus of Fränkel could cause primary broncho-

pneumonia. His results have been confirmed by Cornil, Babes, and Neumann, the latter of whom found the pneumococcus in cases of primary bronchopneumonia. Quesiner and Neumann found the pneumococcus in the sputum of children suffering from bronchopneumonia.

The secondary form of bronchopneumonia may be caused by streptococci (Northrup and Prudden), which invade the lung-tissue from the trachea, as in diphtheria. Guarnieri also found streptococci in the lungs of children dying with bronchopneumonia after measles. On the other hand, these secondary types of bronchopneumonia may also be caused by the pneumococcus of Fränkel, which causes the primary type of the disease. This has been shown in the work of Netter on the subject, and confirmed by Banti, Strelitz, and Baginsky. In diphtheria the Klebs-Löffler bacillus may be found in the lung areas of secondary bronchopneumonia (Babes, Frosch, Baginsky). The Eberth bacillus has been found in areas of bronchopneumonia complicating typhoid fever (Polyniere).

**Morbid Anatomy.**—The essential lesion in bronchopneumonia is an inflammation of the walls of the bronchi and of the air-spaces surrounding the inflamed bronchi (Delafield). The walls of the bronchi are thickened and infiltrated with small round cells; those of the alveoli of the lung are thickened and their cavities filled with fibrin, pus, epithelial cells, and new connective tissue. The smaller bronchi are dilated and contain pus, their walls being infiltrated. The inflammation may also be conveyed from the bronchi to the parenchyma of the lung by aspiration of secretion (Ziegler). In the latter case the smaller bronchi are occluded, collapse of the lung follows (atelectasis), and a pneumonia thus results. On section there are seen grayish-red, gray, or yellowish-gray areas of varying consistency, which correspond to a cut bronchus and its surrounding peribronchitic pneumonia. If the areas are croupous, they have a more granular appearance. Small areas of this form of pneumonia may coalesce, and thus whole lobules of the lung be consolidated. These larger areas may be separated by lung-tissue which contains air, or a whole lobe may become consolidated, as in lobar pneumonia. The exudate found in the affected alveoli is at first composed of desquamated swollen epithelial cells, and later of leucocytes. If the exudate has a more fluid character, it is called catarrhal. It then contains more serum than fibrin. If the fibrin is in excess, the exudate has greater consistency, resembling that in lobar pneumonia, and is then called croupous. The catarrhal and croupous forms of exudate may both exist in a lung affected with bronchopneumonia. Blood-cells may predominate in the exudate, so that the lung may on section have a hemorrhagic appearance. This is apt to be the case in streptococcus inflammation and also if foul fluids have been aspirated.



The mucous membrane of the bronchi is the seat of catarrhal inflammation.

There is inflammation of the pleura to a varying degree.

The bronchial and mediastinal lymph-nodes may be enlarged with simple or tuberculous inflammation. There is œdema of the lung tissue which is not inflamed. Bronchopneumonia may result in resolution and restoration to the normal. Suppuration and formation of abscess with destruction of lung tissue, or gangrene of the lung, may result in rare cases.

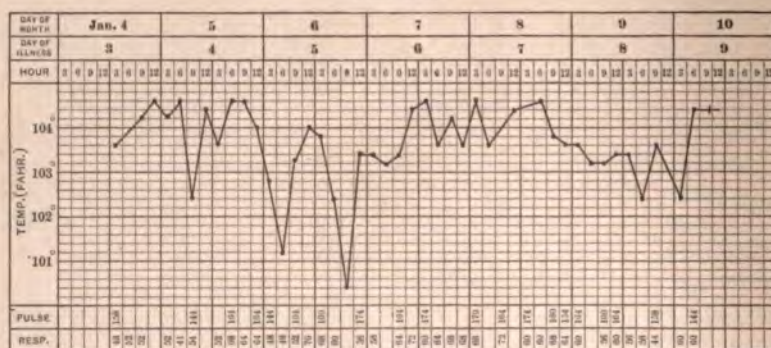
Persistent bronchopneumonia in children results in induration of the lung. There is an increase of the connective tissue of the alveolar septa, of the walls of the smaller and larger bronchi, and also of the walls of the peribronchial vascular tissue. The lung on section is seen to be studded with fibrous nodules, or a whole lobule or lobe may be converted into connective tissue.

**Symptoms.**—Bronchopneumonia is divided clinically into several distinct types. In newly born and very young infants the disease may set in insidiously. The infant is born in good condition; after some little exposure it develops slight snuffles and a slight cough. Dyspnoea then appears. All this may occur within the first eight days after birth. The cough becomes more harassing and the dyspnoea more marked. Slight cyanosis supervenes after a time. The infant is restless and does not sleep, the cyanosis becoming more marked and constant. The infant may have frequent convulsions. The dyspnoea finally becomes so marked as to cause distinct drawing inward of the lower part of the chest-wall with each inspiration. In these cases there is little or no temperature; in that respect they resemble cases of bronchopneumonia in extremely old people. The temperature may be slightly subnormal even when the infant is mortally ill with a disseminated bronchopneumonia. The cough may not be marked. These cases should be differentiated from those occurring in infants born with an atelectatic condition of the lungs. In the class of cases under consideration, atelectasis develops as a sequence of the bronchitis and bronchopneumonia. The movements are greenish, containing undigested curds. The infants may finally develop enteritis. The course of the disease is in these cases very acute. The infant either rapidly grows worse or begins to improve immediately. The former course is, however, the rule in this very dangerous and insidious form of bronchopneumonia. If the infant does not improve, the cyanosis becomes more marked, as does also the dyspnoea; the respirations increase to more than 80 a minute, the pulse becomes very rapid, and the heart feeble; the infant lies in a soporose state; the end may supervene with tympanites, convulsions, and œdema of the lung. This form of bronchopneumonia is very frequently overlooked at the outset and mistaken for a simple bronchitis.

Another form of bronchopneumonia in infancy begins as a simple bronchitis, and may be treated as such for days. Finally, posteriorly in both lungs there are found the fine crepitations which give warning of the presence of bronchopneumonic processes. Bronchopneumonia of this variety runs its course without temperature. It occurs in rachitic or weakly infants and children, or follows a mild attack of influenza. The attacks of coughing are especially troublesome, and are frequently followed by vomiting of the contents of the stomach. The movements are loose, and show greenish particles and undigested white flaky masses. The dyspnoea is constant and characteristic, and if the patient is out of bed, grows more marked toward the late afternoon. The *alæ nasi* are dilated. The temperature rarely rises above  $101^{\circ}$  F. ( $38.3^{\circ}$  C.), and is generally  $100^{\circ}$  F. ( $37.2^{\circ}$  C.) or even lower. The course is favorable; the cough may persist for weeks after the subsidence of the acute symptoms, being especially marked at night.

A more common form of bronchopneumonia in infancy begins as a simple bronchitis, which may last for a few days, when, without warning, the infant has a chill followed by a rise of temperature, the case having suddenly developed into a full bronchopneumonia. In a six weeks' old infant with disseminated patches

FIG. 106.



Fatal bronchopneumonia; reinvasion of the lung on the fifth day. Infant, six months old.

of pneumonia, the chill was so severe as to cause extravasations of blood underneath the surface, with markings resembling those seen in marbling of the surface. In another case the chill was so severe that an immediate fatal issue was feared. In that bronchopneumonia sometimes begins with a chill, it resembles a lobar process.

The most common type of bronchopneumonia may begin with a rise of temperature preceded by vomiting. The harassing cough



is present from the outset, causing the patients to cry with pain at each attack. There is no sputum, but in very young infants a frothy mucus may in the later stages of the disease collect about the mouth. The dyspnoea is marked. The alae nasi are dilated at each inspiratory effort. The peripneumonic groove is depressed, and in very severe dyspnoea the suprasternal region may also be depressed at each inspiration. Very frequently the dyspnoea will resemble that due to laryngeal stenosis. There are, however, none of the signs of laryngeal obstruction, such as laryngeal breathing.

**Fever** is always present in infants and children, except in the classes of cases above noted. It may reach  $106^{\circ}$  F. ( $41.1^{\circ}$  C.), and is as a rule remittent. It may fall gradually to the normal, and in the favorable cases may reach the subnormal and remain there for a few days. The course of the fever is, however, not an indication

FIG. 107.



Ordinary type of bronchopneumonia. Recovery. Female child, one year and six months of age.

of the severity of the disease. Fatal bronchopneumonia sometimes shows a steady decline in the temperature toward the approach of the fatal issue. In other cases the temperature may drop to the normal, remain there a few hours or a day, and then rise sharply to  $104^{\circ}$  F. ( $40^{\circ}$  C.) or higher, thus indicating that a new part of the lung has been invaded by the disease (Fig. 106). Such rises of temperature after a fall to the normal are of grave import if they occur in an infant acutely ill with a process which has been severe for days. They show a tendency of the process to spread, and in young weakly infants such an extension of the process is apt to be fatal. A drop by lysis to a normal temperature which continues for a few days, and is followed by a slight gradual rise with subsequent remissions to the normal is also common, and may indicate a return of the bronchopneumonic process, or a pleuritic

effusion of a purulent character. The physician should be on the alert for an effusion in the cases which have run an irregular or remittent temperature for a period of more than two weeks. I have, however, operated upon cases of empyema following bronchopneumonia in infants, in which the temperature-curve was normal for days, and then showed occasional rises to  $101^{\circ}$  or  $102^{\circ}$  F. ( $38.3^{\circ}$  or  $38.8^{\circ}$  C.).

The pulse is as a rule rapid. It is difficult in infants to estimate its exact character. It is, however, always possible to distinguish the abnormally weak and thready pulse even in the youngest infant. The rapidity of the pulse varies widely even in the favorable cases. Its ratio to the respiration (the pulse-respiration ratio) is, as a rule, maintained in favorable cases, but is not always so. Even if it be so much distorted as to present the ratio of 1 to 2, the patient may make a good recovery. The character of the pulse and respiration should therefore be judged in connection with other signs of decreasing heart power, such as abnormal pallor, coldness of the surface, and cyanosis however slight. In artificially-fed infants who are above the average weight, the beginning of cardiac weakness is indicated by an abnormal pallor of the face and slight cyanosis of the lips.

**Sputum.**—In young infants there is no sputum, nor is it probable that in uncomplicated cases of bronchopneumonia the younger infants cough up and swallow sputum, as is generally supposed. At most, there is after severe attacks of coughing a collection about the lips of frothy mucus probably coming from the trachea.

**Gastro-enteric Tract.**—The symptoms referable to the stomach and intestine are of great importance in severe bronchopneumonia of the primary type. Even up to the second year of life tympanites sets in very early. In one case it was so distressing a symptom as to mislead the physician into thinking that peritonitis might be present. It is especially apt to set in with rachitic and weakly infants who have been fed on the bottle. If it appears late in a very sick infant, it is a symptom of grave import, and may sometimes cause the fatal issue. In some cases the pre-agonal distention is very extreme, and so far as can be judged very painful. Some infants begin to vomit from the outset of the pneumonia. The vomiting may occur once or twice in the twenty-four hours, or may be incessant. With the vomiting there may be the passage of greenish stools or a fully developed enteritis of severe type. So severe is the enteritis in some cases as to cause the death of a patient suffering from pneumonia of only moderate severity. This form of the disease does not occur exclusively in the summer months, but is more prevalent at that time.

**Cerebral Symptoms.**—The infant is in some cases stupor from the outset of the disease. Older children may have slight convulsive



twitchings of the muscles of the face and extremities. In cases in children at the third year there may be complete unconsciousness and symptoms simulating those of meningitis, such as rigidity of the muscles of the neck. I have seen the cerebral symptoms persist for weeks in young infants who made complete recoveries. In other cases in young infants and children, the bronchopneumonia may partly resolve, and still there may be a continuance of the cerebral symptoms or even an exacerbation of them. In these cases the possibility of the presence of otitis or mastoid inflammation should be seriously considered.

The secondary form of bronchopneumonia may complicate the exanthemata—measles, scarlet fever, varicella, typhoid fever, pertussis, influenza, and diphtheria, and also gastro-enteritis or any form of infection, such as that of septic wounds or osteomyelitis.

The symptoms of bronchopneumonia which complicates **pertussis** are of an unequivocal character. A febrile movement may be present with a simple bronchitis. If bronchopneumonia is imminent or present, the fever is marked and constant, and may reach  $106^{\circ}$  F. ( $41.1^{\circ}$  C.). The dyspnoea is very marked, but the cough may not be increased. In certain forms of pertussis without complications there is a slight constant dyspnoea, which is due to the disease. If bronchopneumonia is a complication the dyspnoea is more decided, the number of respirations three or four times the normal, and the pulse-rate increased. There is marked cyanosis. There may be all the symptoms of a severe bronchopneumonia, such as tympanites, vomiting, and green diarrhoeal stools. The bronchopneumonia is, as a rule, of the disseminated type, with areas of consolidation of greater or lesser extent in both lungs. The infants are much more ill than they would be with a primary process of the same extent. A bronchopneumonia of this kind can be diagnosed if upon examination of the chest there are, in addition to the physical signs of bronchitis, fine crepitations over the different parts of the chest, especially over the lower lobes of both lungs posteriorly. There may also be dulness with bronchophony and bronchial breathing over small areas, either in the upper or lower lobes of the lung on one or both sides. The bronchopneumonia of pertussis may supervene at any period of the disease, and is not the result of exposure. On the contrary, it may occur in infants and children who have been most carefully protected from exposure. It is the result of the form of disease—a mixed infection. The pertussis probably makes the lung more viable to disease in some subjects than in others. The bronchopneumonia is a grave complication, and is very fatal. It may cause complications, such as pleurisy of a serous or purulent nature, and often opens the way for invasion of the lung by tuberculosis. It may run a chronic course (persistent pneumonia) and reduce the patient to a very weak state. The patient will then

develop consolidation of a whole lobe of the lung which will take weeks to clear up.

Bronchopneumonia complicating **measles** supervenes, as a rule, in the stage of eruption, and is a very serious complication. Its presence may be suspected if, on examination, of the chest, there are found, in addition to the râles of bronchitis, very fine crepitant râles over areas disseminated through both lungs. This complication also causes a febrile movement after the fading of the eruption. There are severe cough and dyspnoea. The pulse may reach 180 to 190, and the respirations 90, but the patient may recover even if the signs of cardiac weakness, such as cyanosis, are marked. The patient is stupid, does not take food or notice his surroundings. Sometimes there may be other signs, such as hemorrhages into the eruption (so-called hemorrhagic measles), indicating that the process is one in which there is a mixed infection. There may be a complication of serous or seropurulent pleurisy.

Bronchopneumonia complicating **typhoid fever** does not, as a rule, give very striking features apart from those belonging to the latter disease. It seems to be of a mild and insidious character. The bronchopneumonia of typhoid fever is apt to mask the typhoid if it appears at the outset of the disease. There is then a typhoid beginning as a pneumonia. The area of bronchopneumonia is well localized. It may be a small area in the upper or mid-region of the lung. The febrile curve in these cases may range quite high at the outset and thus mislead the physician. The process persists for weeks, sometimes as long as five weeks. The lung is slow in clearing up. The signs of dullness, bronchial voice and breathing may persist into convalescence. In other cases the pneumonia may supervene in the course of the disease. It can then be detected only if the cough is harassing and the dyspnoea marked. In delirious patients the pneumonia can only be discovered by repeated and constant examination of the chest. These cases are not so apt to develop pleurisy of a serous or purulent nature as the pneumonia complicating measles or scarlet fever.

**Varicella** is only rarely complicated by bronchopneumonia. In this disease also the pneumonia runs a protracted course, but is less serious in its outcome than in the other exanthemata. It occurs in the severer forms of varicella in which the eruption is complicated with abscesses or necrosis of the skin (mixed infection).

**Scarlet fever** is not so frequently complicated by bronchopneumonia as measles, but when it does occur the bronchopneumonia is of a very severe type. It occurs in the septic forms of scarlet fever, and may appear early in the disease, on the fading of the eruption. Scarlet fever complicated by bronchopneumonia is frequently followed by pleurisy of a purulent nature.

The bronchopneumonia which complicates **diphtheria** has been



carefully studied by Northrup and Prudden. It is the result of a streptococcic invasion of the lung or an invasion by the Klebs-Löffler bacillus. As a rule, however, it is a mixed infection, as was pointed out by Northrup and Prudden. The laryngeal form of diphtheria frequently proves fatal through this complication.

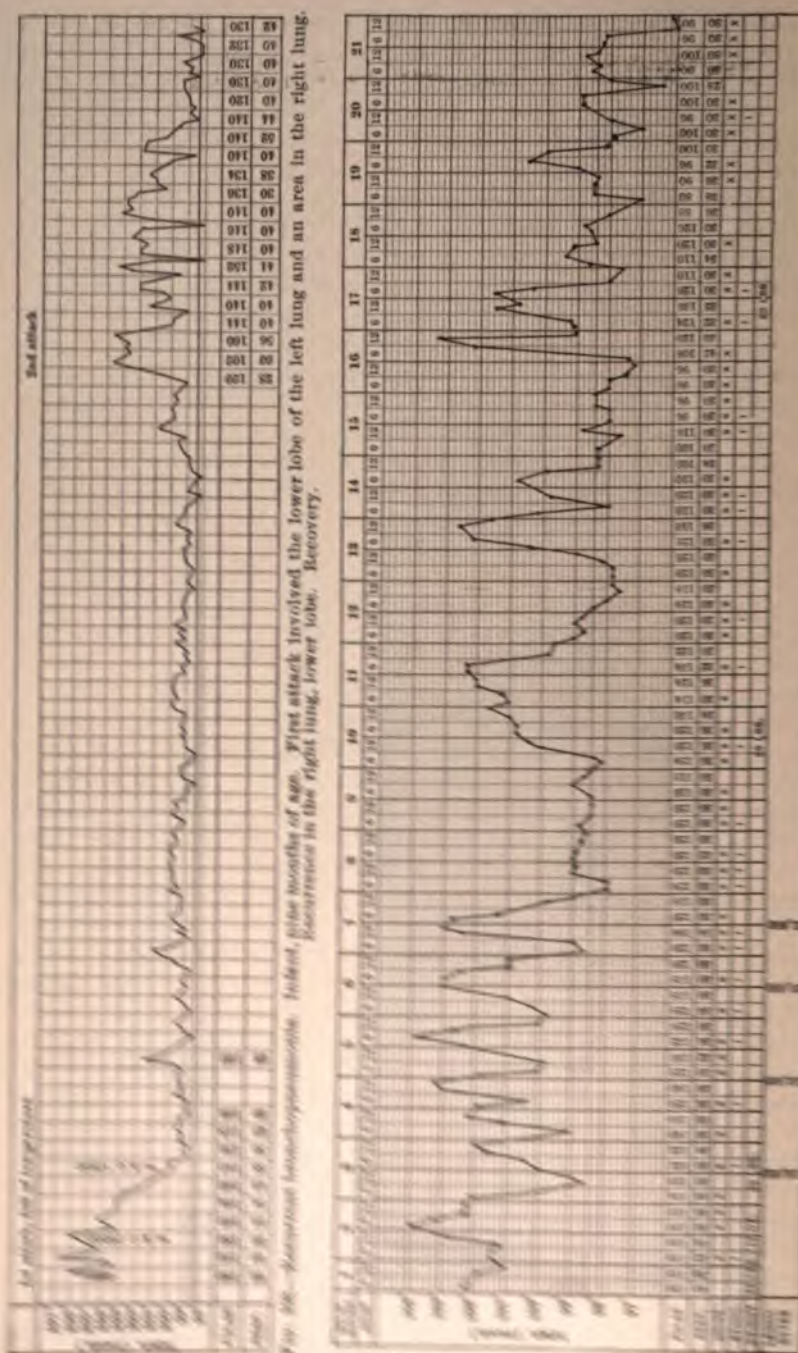
Of special interest is the bronchopneumonia which complicates chronic or subacute **diarrhœal conditions**. This form, which is of a distinctly septic type, is caused by infection of the lung by streptococci, which invade the general circulation through erosions in the mucous membrane of the gut (Booker, Czerny, Fischl). It is not always due, as was formerly supposed, to keeping the infant in the recumbent posture, nor does it occur in hospital practice alone, but is frequently seen in private practice in infants in unhygienic surroundings. It is of the persistent type, and runs its course with a daily high or low febrile curve, and results in areas of consolidation, which sometimes involve a whole lobe of a lung. This form of pneumonia is one of the fatal complications of the subacute intestinal catarrhs.

Some infants, after one attack of bronchopneumonia, have repeated or recurrent attacks on the least exposure (Fig. 108), in some cases developing catarrhal croup. In other cases, there develops an emphysematous condition of the lung, in which the least exposure or change in the atmosphere will cause an asthmatic attack.

**Course, Termination, and Complications.**—Bronchopneumonia may terminate in complete recovery and restoration of the lung to the normal, or may prove fatal. The mortality varies at different times and with the environment. The prognosis in marantic infants, and also in bottle-fed infants, is very bad. Rachitic infants have bronchopneumonia with a very protracted course (Fig. 109). The forms which complicate measles, pertussis, scarlet fever, and influenza are very fatal. Abscess or gangrene of the lung may be a complication. In some forms of otitis the symptoms may very closely simulate those of tuberculous meningitis. Otitis prolongs the disease and frequently misleads the physician. Especially trying are the forms of bronchopneumonia of very limited extent in one or both lungs, in which, after the disease has run its course, there is a protracted, remittent or intermittent fever-curve. Serous pleurisy and empyema are very common complications. Their presence may be suspected if the disease runs a course protracted beyond two weeks, and if signs, such as dulness, flatness, and bronchophony, persist and become more marked over the whole side of the chest.

**Meningitis** may complicate the disease. Care should be taken not to confound cerebral symptoms with true meningitis.

**Pericarditis** complicating bronchopneumonia is apt to be purulent, and is rarely diagnosed during life. I have seen cases in





which during life repeated examinations failed to reveal positive signs of effusion into the pericardium, but in which purulent pericarditis was found at autopsy. This is frequently true of cases in which the effusion is limited (30–50 grammes). If bronchopneumonia occurs in the left lung with consolidation anteriorly and some pleural effusion, it is almost impossible to diagnose moderate pericardial effusion. The complication is very fatal.

**Physical Signs.**—Clinically the physical signs of bronchopneumonia are divided in those of the following stages: the first stage—*invasion*, the second stage—*consolidation*, the third stage—*resolution*. There is no sharp line of demarcation between the signs of the stages.

**First Stage.**—Inspection shows the face to be flushed on one or both sides, and the nostrils to be dilated; with each inspiration there is drawing inward of the peripneumonic groove and sometimes of the suprasternal tissues over the upper part of the trachea.

**PALPATION.**—If bronchitis is present, there may be rhonchal fremitus, but it is frequently absent.

**PERCUSSION.**—In the early stage, there is, just before consolidation, slight dulness over small areas, which in young infants with thin-walled chests may have a slightly tympanitic note (tympanitic dulness). Other parts of the chest may have a vesiculotympanitic note.

**AUSCULTATION.**—If bronchitis is present, the râles of bronchitis may be heard. The respiratory murmur is rude. By careful examination of all parts of the chest one or more areas in which are heard fine crepitant râles may be found. They may easily be overlooked, and may disappear when the infant cries or coughs, and during the examination.

**VOCAL RESONANCE** is slightly increased over areas in which there is slight dulness or the beginning of consolidation. The whole posterior aspect of the thorax from above downward, and also the axillary fossa, should be examined. The apex of the lung in front, and the lower part of the thorax in front and behind on both sides, should be carefully examined, as well as the areas of the borders of the lungs where they come in contact with the chest-wall. Increased vocal resonance and slight dulness alone, especially over the apex of the right lung in front and behind, should be accepted with great caution as indicative of the beginning of bronchopneumonia.

Dyspnoea should not be looked upon as a sign of pneumonia. The crepitant râle in a circumscribed area or in several areas is the sign pathognomonic of this stage.

**Second Stage.**—**INSPECTION** shows no condition differing from those of the first stage.

**PALPATION.**—If the area of consolidation is limited, there is no change, because the area and the chest are small. If there is effusion in the lower portion of the pleural cavity, the fremitus may be dimin-

ished over the lower part of the chest, although the pneumonia is in the upper part. *Fremitus* is therefore misleading, and is only confirmatory in the presence of other signs.

**PERCUSSION** reveals dulness in complete consolidation or dulness with a tympanitic note in the beginning of consolidation, and also flatness if fluid is present over the consolidated area in the lower part of the lung. The dulness may involve a very small area or an entire lobe of the lung. There may be slight resistance to the percussing finger over the consolidated area. The unaffected lung is hyperresonant.

**AUSCULTATION** gives bronchophony and bronchial or bronchovesicular breathing over the consolidated areas. These are not necessarily present over consolidated lung. In infants and children there may only be abnormally rude respiratory murmur and increased vocal resonance. Fine crepitant pleuritic râles may be heard over the consolidated area.

*Diagnostic stress* is to be laid on complete dulness with bronchophony and bronchial breathing.

**Third Stage.**—**PALPATION** will give increased *fremitus* if the area of lung consolidation is large and there is no fluid over the area.

**PERCUSSION.**—As in the first stage, there is dulness to a varying extent, with a tympanitic note showing the return of air into the lung.

**AUSCULTATION** gives a crepitant râle, as in lobar pneumonia. The voice and breathing are less bronchophonic. Dulness may persist for days or weeks. In some cases there is fluid, which increases the dulness or flatness. Dulness, crepitant râles, bronchophony, and bronchial breathing are constant features, and are diagnostic. In infants and children, bronchophony is more constantly present than bronchial breathing. In the bronchopneumonia of the newly born infant, it is sometimes possible to discover with the small bell of a stethoscope areas in which air does not enter (atelectatic).

**Equivocal Signs likely to be Mistaken for the Beginning of Bronchopneumonia.**—In infants and children, the physician is apt to be easily misled into a diagnosis of incipient bronchopneumonia. Equivocal signs—*i. e.*, signs which are not absolutely diagnostic—are apt to be met in certain parts of the chest and in the presence of rational symptoms, such as fever or apparent dyspnoea, undue importance may be attached to them. These signs are as follows:

*a.* A slightly high note on percussion and an increase of vocal resonance or *fremitus*, with a rude respiratory murmur on the right side over the apex in front or behind. It should not be forgotten that this region, especially in infants, normally shows varying degrees of these signs as compared with the left side.



b. A slight dulness over the lower part of the chest on the right side behind, due to the presence of the liver, is normal. To be abnormal, the dulness must be very marked and the vocal resonance much increased. The resistance to percussion must be pronounced in order, in the absence of more positive signs, to justify a suspicion of the beginning of consolidation.

c. Bronchial or bronchovesicular breathing too near the vertebral column behind on either side, between the scapulæ, should be cautiously interpreted. In some infants, the breathing in this region is normally bronchovesicular. It is in this region that the diagnosis of *central pneumonia* is so often made—a diagnosis rarely verified by the subsequent course of a case.

d. In some infants and children, especially from six to ten years of age, it is found that the fremitus and vocal resonance diminish behind from a short distance below the angle of the scapula to the base of the lung; the breathing also is heard less distinctly. A diagnosis of incipient pneumonia or consolidation with fluid requires positive and unmistakable evidence very low down behind. The thick muscles of the back and organs behind the thorax, such as the kidney and liver, obscure slight signs below the ninth or tenth rib. Slight variations from the normal should not receive undue attention.

**Diagnosis.**—Bronchopneumonia should be differentiated from the lobar fibrinous form of the disease. In children above five years of age this is not difficult; in those under the second year, in whom fibrinous or lobar pneumonia is not uncommon, a positive diagnosis of lobar pneumonia cannot be made until the stage of consolidation, and even at that time only as to distribution. In the main, it is made from the course of the temperature. In lobar pneumonia the temperature will fall by crisis after the usual period. A marked leucocytosis, which increases toward the day of crisis and then rapidly diminishes, is also a characteristic feature. There should be also the physical signs of lobar consolidation.

If these symptoms and signs are all present, it may be assumed *clinically* that a lobar pneumonia is to be dealt with. Such a diagnosis is always open to doubt, for a bronchopneumonia may have the lobar consolidation and the leucocytosis, but will rarely have the critical drop of temperature which occurs in lobar pneumonia. As to the onset, bronchopneumonia may set in with a chill, and lobar without one. The complications in both forms are identical; empyema is as likely to occur in one as in the other. Lobar pneumonia is rarely prolonged in duration if complications are absent, while the bronchopneumonic type of disease is, as a rule, of longer duration and may be prolonged into a chronic course.

Disseminated patches of consolidation in a lung in which there is general bronchitis point to bronchopneumonia; diffuse bronchitis,

with fine crepitations in the lower lobes of both lungs, to bronchopneumonia. The presence of a primary disease—measles, scarlet fever, typhoid fever, and influenza—will also influence the process in the lung. The secondary pneumonia is a bronchopneumonic process.

**Prognosis.**—The mortality of bronchopneumonia, even under the favorable conditions of private practice, is as high as 25 per cent. In hospital practice it is much higher, and may reach 50 per cent. or more. It is increased in bottle-fed, rachitic, prematurely born, and syphilitic infants, and is greatest in the first year of life. The disease is especially fatal in newly-born infants, and in cases of gastro-intestinal disorder. The mortality rate increases in New York City in the months of December, January, and February, during which the weather is alternately moist, warm, and cold. Certain years show an increased mortality because of the severe nature of the epidemic.

At the bedside, a prognosis is based on the condition of the lung, temperature, heart, and the presence or absence of nervous symptoms. A persistently high temperature, if there are areas of consolidation in both lungs, is of serious import. An abnormal pallor or slight cyanosis in a bottle-fed baby, even if well-nourished, is a danger signal. Forced and irregular action of the diaphragm is serious; marked drawing inward of the sides of the chest, sometimes as high as the eighth rib, is a very unfavorable sign in infants. These cases show a depression of the suprasternal notch as marked as that which occurs in laryngeal obstruction. Repeated convulsions and jaundice, with enlargement of the spleen, in rachitic infants indicate intense toxæmia. These cases are fatal. Marked tympanites at the end of the first week, in connection with diarrhœa and weakness of the heart, is an unfavorable symptom. Dyspnoea with respirations irregular in rhythm and depth denotes diffuse involvement of both lungs, and is present in the unfavorable cases. Cerebral symptoms supervening late in the disease are unfavorable.

The favorable signs are a good muscular quality of the first sound of the heart, red lips and warm surface, good reaction after hydrotherapy, and periods of quiet sleep with full noiseless breathing, movements of the bowels normal or slightly green, and an absence of marked tympanites. Caution should be exercised in making any prognosis in a bronchopneumonia which shows a marked tendency to involve new areas of lung.

In the **treatment** of bronchopneumonia of infants and children, it should be borne in mind that the disease is a self-limited, acute, infectious one, and that there is no remedy which can abort it or prevent complications. As in lobar pneumonia, the ill effects of the disease must be counteracted as much as possible and the strength of the patient supported. Since the patients are



of very tender age, remedies which are powerful in their ultimate effects are to be carefully avoided. The indications in the treatment are to counteract the effects of the temperature and to support the heart.

The **temperature** in the most fatal forms of this disease in newborn infants is below the normal at times, and rarely reaches a very high point. In other cases of bronchopneumonia in older infants and children, it remains persistently above 103° F. (39.7° C.). In these cases, as in lobar pneumonia, the various forms of hydrotherapy are utilized. Of all the methods, the cold compress applied to the chest, as before described, seems to be the most efficacious. Compresses lower than 70° F. (21.1° C.) are not applied. The applications may be renewed every hour, if the patient bears them well. It sometimes happens that a compress wrung out in water at 70° F. (21.1° C.) will depress the patient, causing cyanosis without reaction. In such cases, as in the lobar cases, I have found the warm bath, 105°-107° (40.3°-41.6° C.), of the greatest utility in relieving the nervous symptoms, such as restlessness and convulsive twitchings. Infants, as a rule, will not bear baths below 80° F. (26.6° C.). I therefore do not utilize the cold full bath in bronchopneumonia in infants. I do not think it advisable to use the bath at 90° F. (32.2° C.) or higher, with cold douching of the head and shoulders, to obtain reaction in infants. The procedure rouses the patients only momentarily, and the subsequent depression is greater. Cold packs over the whole body are also heroic remedies, but are advocated by some authors.

The **heart** is supported by means of digitalis, strychnine, camphor, musk, caffeine, and ammonium carbonate. Of these agents, the most useful are digitalis, strychnine, and musk.

*Digitalis* is administered in the form of the tincture. A drop is given for every six months of the age of the patient. It should not be used unless the pulse is high, and should then be given every three hours. It is discontinued after being administered for two or three days. The effects of stronger preparations, such as the fluid extract, cannot be gauged so carefully as those of the tincture, and they are therefore less useful. The cases in which digitalis is of the greatest value are those in which there is cyanosis to a mild degree, or excessive pallor denoting great cardiac weakness.

*Strophanthus* may be administered alone or in combination with digitalis. The tincture is the form generally used.

*Strychnine* is one of the most useful drugs in the treatment of the heart. An infant six months old will bear grain  $\frac{1}{250}$  or  $\frac{1}{200}$  (0.0003 or 0.00025) very well. Older infants and children bear grain  $\frac{1}{150}$  (0.0004) quite well. Strychnine should not be used in cases where there is excitability of the nervous system.

Atropine, which is so useful in adults, is not well borne by infants and children.

*Ammonium carbonate* is one of the most useful drugs when for any reason *digitalis* cannot be used. Convulsions or restlessness are treated with the bromides of potassium and sodium, which may be combined. Chloral hydrate is combined with both, especially where one dose of bromide of potassium and chloral hydrate is given per rectum.

I do not use poultices. Some authors use them as a routine measure.

*Inhalations* of benzoin and turpentine are of little efficacy. They do not affect the local lesion in the lung, nor do they act on the mucous membrane as they do in catarrhal processes of the nose and throat. In some cases I have seen harm result from overloading the atmosphere with the odor of balsams.

The patient should be isolated from the healthy children of the family and the room kept at a temperature of from 68° to 70° F. (20° to 21.1° C.) and well ventilated. An open wood fire is the most satisfactory method of heating and ventilating the sick-room.

In threatened cedema of the lungs I have found, as in lobar pneumonia, that the right ventricle is best relieved by nitroglycerin, grain  $\frac{1}{200}$  to  $\frac{1}{100}$  (0.0003 to 0.006) being given at a dose, and by the constant administration of oxygen containing 20 per cent. of nitrous oxide.

Whiskey is so universally used that the mode of administering it should receive special mention. Alcohol should not be used as a routine remedy. In some of the milder cases its use is superfluous. There are other cases in which its use must be suspended because of the constant vomiting. In the severer types of bronchopneumonia, in which the temperature is persistently high, the effects of the toxæmia may be counteracted by administering whiskey. Infants receive from minims xx to xxx (1.2 to 2.0); older children a drachm (4.0) every three hours. The whiskey should be well diluted, and should be given after the nursings.

The feeding of infants who take a substitute for the breast should be carefully watched, especially in bronchopneumonia, a disease in which diarrhoea is apt to supervene. If diarrhoea is present, the milk should be discontinued and a cathartic given. The infant is given a high rectal injection of warm normal saline solution twice daily, and is kept on solutions of egg-albumin and acorn cocoa until the intestinal symptoms subside. Milk is then again given. In these cases of intestinal disorder it is of the utmost importance to see that the milk is fresh and uncontaminated.

The cases not complicated by diarrhoea are given a warm high rectal enema of the normal saline solution once daily. In infants,



this procedure will ward off tympanitic distention of the abdomen and stimulate the heart.

The cough is sometimes very harassing, and then only should be relieved. The camphorated tincture of opium or the wine may be given in moderate doses. Codeine is useful in older children; morphine should not be used. In the many hundreds of cases which I have treated I have not found it necessary to use it. Strapping the chest to relieve pain is harmful in infants and children. The chest in these subjects is resilient, and any limitation of its action reacts unfavorably in preventing a full expansion of the unaffected lung.

### PERSISTENT BRONCHOPNEUMONIA.

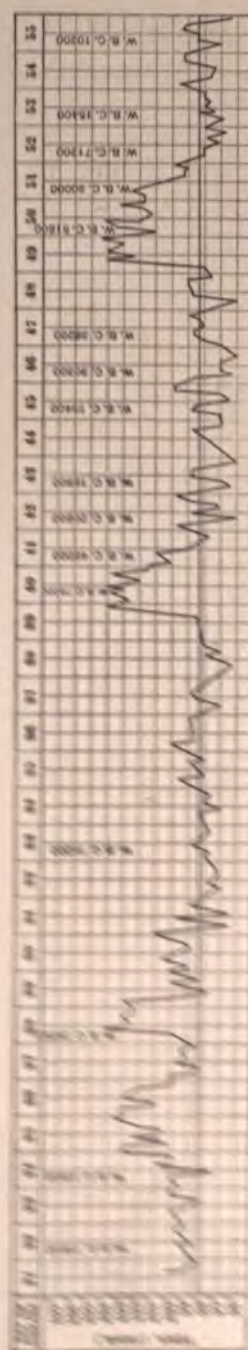
(*Chronic Bronchopneumonia.*)

Persistent bronchopneumonia is a distinct type of bronchopneumonia the course of which extends over weeks or months, the patient meanwhile becoming much reduced in flesh and strength. These cases occur in weakly infants, usually in those who are bottle-fed. A distinct type of the disease complicates chronic enteric catarrh. Cases of this class belong in the category of Gastro-Intestinal Sepsis of Fischl, Escherich, and Czerny. Cases of another set complicate and follow pertussis, measles, and influenza. Lastly, there is a true tuberculous form which is not strictly included in the above classification. The condition is thus rarely primary.

**Symptoms.**—The infant or child has at first the symptoms of an ordinary bronchopneumonia. The fever, however, is of longer duration than in cases which recover. Cases of gastro-enteric affection or pertussis will continue to have a remittently high temperature, which may reach  $105^{\circ}$  ( $40.5^{\circ}$  C.), but fall to  $101^{\circ}$  or  $100^{\circ}$  ( $38.3^{\circ}$  or  $37.7^{\circ}$  C.) on the same day. It will remain normal for days, and then rise again, as indicated in the chart (Fig. 110). There are cough, slight dyspnoea, emaciation, and gastro-intestinal disturbances. In cases of enteric catarrh the intestinal disease takes clinically a secondary place. Some of these cases eventually recover in spite of the progressive emaciation and high fever. This is especially the case in persistent bronchopneumonia which complicates pertussis.

**The Blood in Persistent Bronchopneumonia with Recurrent Invasions.**—In the case from which the chart was taken there was a distinct increase of the number of leucocytes with each new rise of temperature and fresh invasion of the lung. The number of leucocytes mounted as high as 80,000 to the cubic millimetre. A differential count showed that the polynuclear neutrophiles ranged at different times from 73 to 82 per cent. of the leucocytes and the small

FIG. 116.



Postulated broncho-pneumonia with focus in the left and the right lung. With each new invasion of the lung there is a marked leucocytosis, which disappears as the temperature falls to the normal. Recovery. Female child, sixteen months of age.

lymphocytes (mononuclear) from 13 to 21 per cent. As the disease progressed, there were also signs of extreme anaemia, microcytes, megalo-cytes, and poikilocytes being present.

**Physical Signs.**—On examination, there are found areas of consolidation of varying extent, generally made out posteriorly over the apex or toward the base of the lung. There are signs of general bronchitis, increase of fremitus, and dullness marked, slight, or combined with a tympanic note. There may be fine crepitations here and there over the chest. If the areas are extensive, there may be bronchophony or bronchial breathing. The complete consolidation of primary bronchopneumonia is not always present. The lung is only partially consolidated, so that the vocal resonance may simply be markedly increased or the breathing may be bronchovesicular.

**Diagnosis.**—Persistent broncho-pneumonia may be suspected if there is an area of dullness at the apex of one lung which does not resolve after a lapse of weeks. In these cases, there are the other signs of partial or complete consolidation at the apices, the base of the lung continuing resonant in the absence of signs of pleurisy. The persistence of fremitus on the affected side, especially in the mid-region of the chest behind, will aid in excluding the presence of fluid. The rest of the lung is in these cases resonant or hyperresonant. In doubtful cases the exploring-needle should be introduced into the chest to ascertain whether fluid is present.

The treatment is practically an extension of the treatment of the primary condition. If there is an affection of the gastro-intestinal tract,



it is treated. If there is pertussis, treatment proceeds on the lines usually followed in that affection. In some cases the administration of iodide of potassium in small doses has seemed to have a beneficial effect on the course of the process in the lung.

## PLEURISY.

(*Pleuritis.*)

Pleurisy in infancy usually occurs as a secondary disease; it is rarely primary.

**Dry pleurisy** is the form in which the pleura is inflamed without any appreciable formation of exudate in the pleural cavity.

**Pleurisy with effusion**, or subacute pleurisy, as it is incorrectly called, is the form in which a serous or serofibrinous effusion is found in the pleural cavity. The form in which the effusion is of a sero-purulent or markedly purulent character is also called empyema.

**Empyema** is therefore a purulent or suppurative pleurisy. There are other forms of pleurisy which occur with neoplasms of the lung or pleurae. These are not discussed in this section.

### Dry Pleurisy.

**Frequency.**—Dry pleurisy, pure and simple, is, in my experience, clinically not common among infants and young children. As an independent affection, it is found more frequently after the fifth year of life. Clinically, the cause of this infrequency in infancy cannot be easily explained. Young infants and children rarely indicate the pain which is the leading symptom. The disease is masked by other symptoms occurring at the same time. Older children locate the pain and direct attention to it.

**Etiology.**—This form may be primary or secondary. As a primary affection it is found in rheumatic subjects, especially those who are or have been subjects of disorders such as endocarditis or fibrinous adhesive pericarditis. In these cases the etiology is the same as that of rheumatism. The condition is secondary to pneumonia. It may be found complicating any of the infectious diseases—influenza, scarlet fever, measles, typhoid fever, or tuberculosis. In such cases the bacterial factor in the etiology is much the same as in the forms which will be considered under Pleurisy with Effusion. Pleurisy may complicate nephritis of the subacute or chronic type. Traumatism will cause this form of pleurisy; exposure to cold or wet will predispose to it.

**Symptoms.**—The cases of simple dry pleurisy not proceeding to the formation of effusion in the pleura, which have come under my notice, gave few symptoms.

**Pain.**—The children in the majority of cases complained of distinct localized pain on exertion or on breathing deeply. There is also some local pain on external pressure. I have seen marked pleurisy of the dry form in which pain was absent. This is most likely to occur in pleurisies secondary to nephritis. In the primary type, the patients continue to walk about, but are pale and have an anxious expression of the face. There is sometimes a rise of a degree or more in temperature and the respirations are increased and superficial. Those forms described by Henoch as setting in with convulsions, high fever, and vomiting, have not in my experience remained dry fibrinous pleurisy, but have proceeded to the formation of effusion in the chest. The duration of dry pleurisy is variable, and in the rheumatic forms may extend over a long period of time.

The **diagnosis** is not difficult, and is made from the physical signs and the history. On examination, a localized area over which there are a large number of dry crepitant râles is found. The râles are heard so close under the ear that they are distinguishable from the crepitant râles of pneumonia. In some cases there is a dry rubbing sound—a pleuritic friction—over the area affected. In the cases without complications there are no other signs. There is little or no dulness and no change in the voice or breathing-sounds.

The **prognosis** is very good. Tuberculous disease of the lung is not a causative agent in these cases in children so frequently as in the adult. The primary dry pleurisies, with proper care, subside and gradually disappear.

The **treatment** of dry pleurisy is very simple. If the subjects are rheumatic, they are put on small doses of salicylate of sodium. The bowels are kept open with a saline cathartic, preferably Carlsbad salts. The patients are kept in bed. It is not advisable to strap the chest to relieve pain. The desired relief can be secured by some local application of iodine or a sinapism. Codeine is administered in moderate doses to relieve the cough and pain.

### **Pleurisy with Effusion (Subacute Pleurisy) and Empyema.**

*(Purulent or Suppurative Pleurisy.)*

**Frequency.**—This form of pleurisy is common in infancy and childhood. The largest number of cases occur before the fifth year (Simmonds). The succeeding five years show the next greatest frequency. Israel found 29 per cent. of 206 cases to be purulent. Mackey estimates the purulent cases at 40 per cent. of the whole number in children, as against 5 per cent. in adults. Combining the statistics of Simmonds and Hofmokl of Vienna, this form is found to have greater frequency in the male sex. According to these authors, the left side is more often the seat of the disease. Simmonds found



the disease to be bilateral in only 7 out of 175 cases. Of 120 of my own cases of empyema, 2 were bilateral. Of these 120 cases, 104 occurred before the fifth year, and 16 between the fifth and tenth years; 39 were between the age of one and two years, and 16 were less than twelve months of age. The youngest patient was two months of age.

**Etiology.**—Primary pleurisy, whether suppurative or serous, is rare. The literature contains cases of acute effusion in the pleural cavity, in which there was apparently no exciting cause or primary lung affection. The etiology must in such cases remain in doubt. Infection may take place through so many avenues that it is difficult to point out the mode of entrance.

Pleuritis, serous or purulent, is generally secondary in infancy and childhood. All forms of lobar or bronchopneumonia may give rise to pleurisy, most of the cases being traceable to this source. The infectious diseases—measles, scarlet fever, pertussis, typhus and typhoid fever, diphtheria, forms of tonsillitis, retropharyngeal and mediastinal abscess, may precede or directly cause an attack of pleurisy. Chronic intestinal sepsis may cause empyema. In the latter case a pneumonia generally precedes the pleurisy or is present at the same time. In sepsis of the newly-born infant, there may be a complicating empyema. Osteomyelitis of the septic streptococcus variety may be complicated by purulent pleurisy. Tuberculous disease of the lung, actinomycosis of the lung, abscess of the liver, abscess in the mediastinum and abscess in the abdominal cavity involving the viscera, may cause pleurisy. Appendicitis may after the formation of abscess cause pleuritis by extension of the process along the coils of gut to the diaphragm. Finally, rheumatism may cause pleurisy of a serofibrinous nature. Exposure to cold and wet is undoubtedly a predisposing cause. In children, it is common to have a history of a fall or a blow occurring just prior to the attack of pleurisy.

**Morbid Anatomy.**—Pleurisies which accompany acute pneumonia are the most frequent. In these, there may be a slight injection of the pulmonary pleura and a loss of the normal lustre. Here and there a few fibrinous threads or adhesions may be found coursing over the surface of the pleura or running from the costal to the pulmonary pleura (dry or fibrinous pleurisy (*pleuritis sicca*)). In other cases, there is a thickened condition of both pleural reflections, caused by the deposit of fibrin on the surface. Sometimes the amount of fluid is small, while the pleura is very much thickened. The pleura itself may be little altered; underneath the fibrin, the lymph-spaces and blood-vessels may be dilated. In some cases there is also a serous or seropurulent exudate containing leucocytes, endothelial cells, and bacteria. The fluid may be clear or bloody, turbid or opaque, yellow or greenish, and thin or creamy in consistency. Large clots

of fibrin may be found floating in the exudate. Adhesions may form pseudo-encapsulations of exudate, binding down the lung and preventing its expansion. In children, however, the tuberculous pleurisy is most likely to cause extensive thickening of the pleura. In addition to the deposit of fibrin on the costal and pulmonary pleura, there is a real inflammatory thickening of the tissue of the pleura itself, with a deposit of tubercle tissue. Serous or purulent exudate is encapsulated by adhesions, while the lung is bound down by layers of inflammatory tissue. In the tuberculous form the changes are progressive. In the acute inflammatory forms, the exudates are absorbed and the fibrinous deposit is organized into new connective tissue. In time the pleura may be restored to the normal. Adhesions, however, form an important factor in acute pleurisy of children. The pleura may in some cases be permanently thickened by a new layer of connective tissue persisting throughout life. There are forms of pleurisy not tuberculous in which this thickened condition not only remains, but extends from the pulmonary pleura into the lung along the interlobular tissue of the lung itself. There are induration and destruction of lung tissue. This induration is seen in connection with persistent bronchopneumonia. The amount of effusion (purulent) is sometimes quite large in children, and may reach 1000 to 5000 cubic centimetres (Simmonds, Hofmökke). In scurvy and morbus Werlhofii, blood may be effused into the pleural exudate.

**Bacteriology.**—Pleurisy or empyema is divided into several groups according to the class of bacteria found in the exudate. It is well established that the bacteria are the essential cause of the disease.

The first and largest group is that in which the pneumococcus of Fränkel, the lanceolate diplococcus, is found. These cases are called metapneumonic. They may occur during the progress of a pneumonia or after it has run its course. In some cases the process in the lung plays clinically a secondary rôle. The pneumococcus seems to occasion very little disturbance in the lung and to spend its force on the pleura. Thus within three days after the initial chill the pleura is filled with serous or seropurulent fluid. Netter found that of 28 pleurisy cases in infants and children 53 per cent. were due to the pneumococcus. In 71 cases of empyema I found the pneumococcus by culture in 49 (69 per cent.).

The second group comprises those cases in which the streptococcus alone, the staphylococcus, or the streptococcus with the pneumococcus or staphylococcus, is found. Netter found that 17 per cent. of his cases were of the streptococcus class; 15 per cent. of my cases were due to this micro-organism. In cases of the septic type, such as complicated sepsis of the newly born or osteomyelitis, or follow scarlet fever, the *Streptococcus longus* is found in the exudate.



These cases are severe. Nine per cent. of my cases were caused by the staphylococcus. In 9 per cent. of my cases of empyema the streptococcus and pneumococcus were both found in the exudate. Although the pleurisy in which the streptococcus and staphylococcus

FIG. 111.

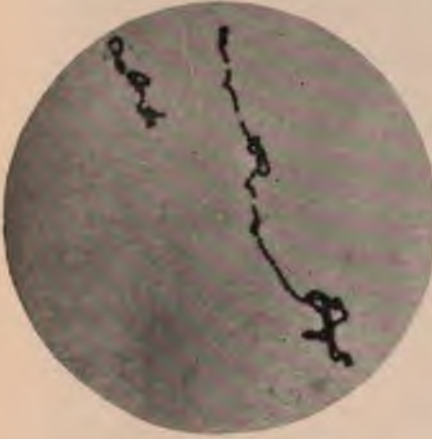


FIG. 112.

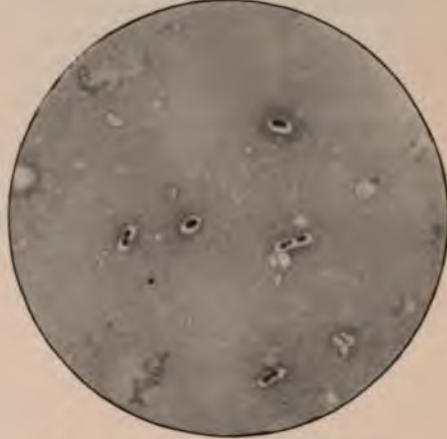


FIG. 113.

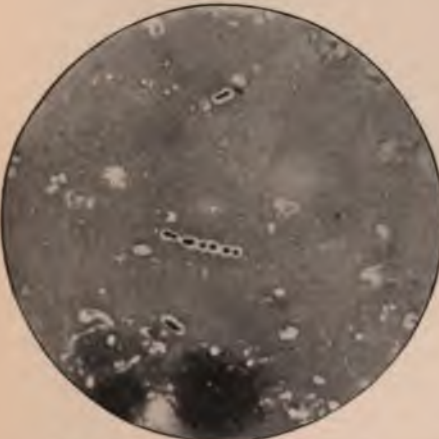


FIG. 114.

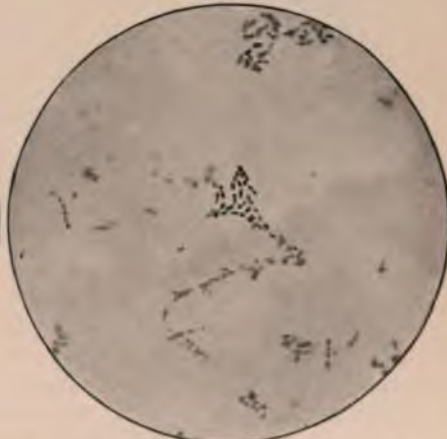


FIG. 111.—Streptococci from the pus of empyema; pure culture.  $\times 1000$ . Photomicrograph.  
 FIGS. 112 and 113.—Pneumococci (*Diplococcus lanceolatus*) from the pus of empyema. Cover-glass preparations showing capsule. Photomicrograph.  $\times 1000$ .  
 FIG. 114.—Pneumococci (*Diplococcus lanceolatus*); pure culture from the pus of empyema. Photomicrograph.  $\times 550$ .

are found may follow a pneumonia, they may also be secondary to a follicular amygdalitis, the exanthemata, typhoid fever, influenza, diphtheria, sepsis, and osteomyelitis.

The third group of cases comprises those in which either the tubercle bacillus is found in the exudate, or the exudate is free from micro-organisms. The latter condition is frequently presumptive evidence of a tuberculous infection (Ehrlich). The tubercle bacillus was found in one of my 72 cases of empyema, while in 3 the findings both by cover-glass spread and culture were negative. This would at most give a frequency of 6 per cent. for the tuberculous variety of pleurisy or empyema.

The last group is that in which micro-organisms other than those mentioned are found in the pleuritic exudate. Such cases have been observed in connection with typhoid fever in which the Eberth bacillus has been found. Escherich has found the coli bacillus in a case of empyema. I have seen one case of this kind. The bacillus of the saprophytic variety and that which causes a putrid empyema are found in cases of this fourth class (Koplik).

The following table shows the relative frequency of the various forms of pleurisy and empyema with the varieties of bacteria in the exudate :

	Children.		Adults.
	NETTER 28 cases.	KOPLIK 72 cases.	
Pneumococcus . . . . .	53.6 per cent.	60 per cent.	17 per cent.
Pneumococcus and Streptococcus . . . . .	3.6 "	9 "	2.5 "
Streptococcus . . . . .	17.6 "	15 "	53 "
Staphylococcus . . . . .		9 "	1.2 "
Putrid . . . . .	10.7 "		
Tubercous . . . . .	14.3 "	7 "	25 "

The most important fact to be deduced from the statistics is that while tuberculous pleurisy in children has a frequency of 6 per cent., adults show a much greater frequency, many of the streptococcus cases being tuberculous in the latter subjects. This figure added to the number of cases in which tubercle bacilli are found in the exudate would bring the frequency in the adult to at least the 45 per cent. given by Bowditch as the relative figure.

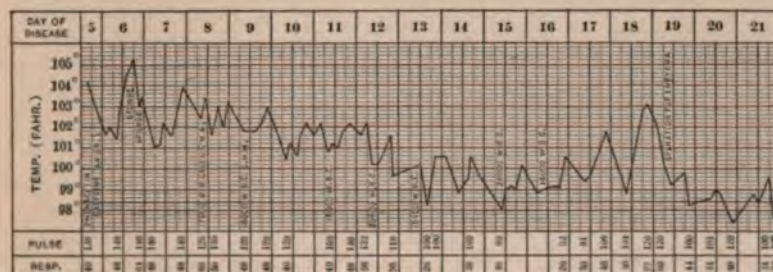
The **physical characteristics** of an effusion in the chest are of clinical importance. An effusion if purulent has usually the gross physical characteristics of ordinary pus. In some cases the effusion is at first clear and serous, but is subsequently seen to be purulent without the occurrence of any extraneous infection. In other cases the effusion may be a cloudy serum, which on exploratory puncture is after a few days found to be purulent. In rare cases the effusion or exudate in the pleura is hemorrhagic. An effusion of that character has not the same significance in children as in adults. In the latter such effusions may be tuberculous or due to some morbid growth of the pleura; this is not necessarily the case in children. I have had a number of cases of hemorrhagic effusion into the pleural cavity. In none of them was there a tuberculous element.



In all, streptococci were found in the effusion, and in some the admixture of blood could be traced to a scorbutic tendency. In one case, in an adolescent with localized effusion of a hemorrhagic nature, there was an actinomycosis of the pleura and lung. The history of this case was not that of an effusion of an acute, but of a subacute chronic nature.

**Symptoms.**—There are no symptoms characteristic or pathogenic of effusion in the pleura or empyema. The condition is in most cases masked by the symptoms of the causal affection. Cases following a pneumonia set in with a chill or a rapid rise of temperature, with which there may be a convulsion followed by stupor or cerebral symptoms. After this onset the fever continues, ranging from  $103^{\circ}$  to  $105^{\circ}$  F. ( $39.4^{\circ}$  to  $40.5^{\circ}$  C.), the pulse being 140 to 180. There will be cough, great dyspnoea, and pain in the chest, which is especially manifest when the infant or child coughs. The breathing is shallow. After a few days the acute symptoms subside, the fever becoming remittent. The temperature may be nearly normal. The dyspnoea continues, although the temperature and pulse may be normal during part of the day.

FIG. 115.



Lobar pneumonia; fall of temperature, by lysis; gradual rise after the thirteenth day, due to empyema. Operation on the nineteenth day. Recovery. Boy, four years of age.

In some of the cases the effusion becomes apparent on the eighth day; in others a purulent effusion is found in the chest on the twelfth or fourteenth day of the disease. The effusion, which finally becomes apparent in the chest, has been coincident in its onset with a pneumonia—there has been a pleuropneumonia. The process in the lung, however, takes a secondary place in the clinical picture when the effusion in the pleural cavity has accumulated.

There is another set of cases in which the course of the disease is insidious. The patient may at the onset have had for two or three days a febrile movement which has subsided, leaving the child not quite well and with a slight febrile movement toward evening, a slight hacking cough, and some little pain in the chest on exertion. Langour and loss of strength are progressive. There may be ex-

hausting sweats at night. Examination of the chest will reveal an effusion.

The metapneumonic pleurisy in infants and children have a characteristic course. The patient has a typical pneumonia. The temperature on the ninth, tenth, or thirteenth day may drop to the normal or subnormal, the respirations continuing high. A gradual rise of temperature follows, with physical signs of fluid in the chest (see Fig. 115). The pulse and respirations rise with the temperature. Toward evening there may at times be chilly sensations. Exploration may discover fully developed effusion in the chest, serous or purulent according to the severity of the pleuritic infection. As a rule the younger the subject, the more likely is the effusion to be of a purulent nature. The duration of the effusion in the chest will also be a guide in determining its nature. An effusion occurring after pneumonia in a young infant and persisting for a week after the pneumonia has run its course, is likely to be purulent.

**Diagnosis.**—There are some symptoms, such as continued dyspnœa, a slight or troublesome cough, exhausting sweats, and a distinctly intermittent range of temperature, which in all cases of pleurisy should direct attention to the chest. None of these symptoms is, however, pathognomonic of pleurisy, serous or purulent,

since they may be found in other pulmonary conditions. The diagnosis of pleurisy with effusion or empyema should take into consideration not only the rational symptoms, but also the physical signs.

The physical signs of pleurisy with effusion and of empyema are identical.

**FLUID IN THE CHEST.**—*A.* The chest partly filled with fluid. *B.* The chest full of fluid.

*A. The Chest Partly Filled with Fluid.*—It is assumed that the greater part of the fluid is in the lower portion of the chest (Fig. 116). In children and infants it does not cause displacement of the viscera.

*Inspection* may show fulness of the lower part of the affected side; the lower part of the chest moves less than the normal side.

*Palpation.*—Vocal fremitus will be felt over the upper portion of the chest in front or behind, and will be lost over the lower portion.

*Percussion* of the chest in front will often give an exaggerated

FIG. 116.



Pleural cavity partly filled with fluid.



hyperresonant tone over the upper lobe of the lung. Behind, there is almost always dulness to a greater or less degree above over the scapula, due either to thickening of the pleura or to an exceedingly thin layer of fluid. This dulness can be distinguished from dulness due to other causes by firm percussion which will elicit the pulmonary note of the underlying lung. Below, over the fluid, the dulness changes to complete flatness.

*Auscultation.*—The voice and breathing may be heard over the whole side with as much intensity as on the healthy side, or with diminished intensity below the level of the fluid. Râles, generally pleuritic crepitations, may be heard above the level of the fluid. Bronchial breathing and voice may be heard over the fluid or at the level of the fluid, but this sign is not absolute.

Diagnosis to justify needle exploration must be based on absence of vocal fremitus over the fluid and its presence above the fluid, dulness behind above the fluid, which on firm percussion gives a faint pulmonary tone and flatness over the fluid with slightly increased resistance to the percussing finger.

*Note.*—The method of examining infants for fluid is invariably that indicated in the earlier part of the book. It is a mistake to examine the infant as it lies in the lap of the mother, for in this position the fluid will gravitate behind. When the infant lies on the face, the fluid will again gravitate to the anterior part of the chest and thus not be made out. In the earlier stages of pleurisy the fluid only partly fills the thorax. On account of the small size of the thorax in infants, it is impossible to determine the change of level of the fluid by changing the position of the patient.

The resonant note over the lung apex in front should, in the presence of dulness behind and flatness below, always arouse suspicion of fluid, for in these cases the lung seems to be compressed upward, forward, and inward, thus causing the vesiculotympanic note in front and above.

The chest is partly filled with fluid, as is shown in Figs. 117 and 118. I have quite frequently found this condition in infants and children who have constantly lain on the back, and in whom adhesions seem to have kept a layer of fluid in the position shown in the figure. It will be assumed for illustration, that the right side is affected:

On *inspection*, fulness of the intercostal spaces on that side may be detected; the movement of the thorax is labored, and the intercostal spaces may be drawn inward on inspiration.

*Palpation.*—Vocal fremitus due to the lung's being in contact with the chest-wall may be present over the anterior aspect of the chest. Posteriorly, the fremitus will be entirely absent.

*Percussion.*—Anteriorly, the note may be vesiculotympanic; posteriorly, there is complete dulness over the whole chest, which is

more marked below. There is rarely the flatness obtained when the chest is full or half full of fluid. There is also resistance to the percussing finger.

By *percussing firmly* the note of the lung beneath will invariably be elicited; breathing-sounds and voice-sounds will be heard as normal or distant.

*Pleuritic crepitations* may be heard over the whole affected side; there is no displacement of the liver or heart on the left side.

*Diagnosis of fluid before exploratory puncture* must rest on the complete or partial absence of fremitus behind, and complete dulness

FIG. 117.



FIG. 118.



Fluid in a thin layer posteriorly in the pleura.

or flatness. The quantity of fluid is small; there is less resistance to percussion than when it is large.

(B.) *The Chest Full of Fluid (Right Side).*—On *inspection*, the objective signs of intense or moderate dyspnoea are found: The chest on the affected side is immobile; the intercostal spaces are retracted with each inspiration; the affected side bulges visibly.

*Palpation.*—Vocal fremitus is lost over the whole side in front and behind. In rare cases a little fremitus is felt.

*Percussion.*—Ordinary and firm percussion give a flat note over the whole chest in front and behind; the resistance to the percussing finger is wooden. In front, flatness may be present over the apex of the lung (Fig. 119). In some cases in children a sound over the apex of the lung, which resembles the cracked-



pot sound over cavities in adults, may be obtained. It may be due to lung compression. In other cases the resonance in front, over the lung of the affected side is vesiculotympanic, owing to the pushing upward and forward of the lung and to its distention.

*Displacement of the Pleural Fold underneath the Sternum.*—A very important aid in the diagnosis of fluid in either side of the

FIG. 119.



Pleural cavity full of fluid. Flatness anteriorly and posteriorly.

FIG. 120.



Pleural cavity filled with fluid. Lung displaced upward and forward. Resonance anteriorly over the apex, either vesiculotympanic or of the cracked-pot quality.

chest is the displacement of the line of the reflection of the pleura in front. Normally the pleurae of both sides meet underneath the sternum in the median line. Above, at about the level of the second rib, they depart gradually from each other. If there is a large amount of fluid in the right chest, the pleural fold of that side becomes distended and displaced to the left, and may be marked out above the heart by dulness to the left of the midsternum. If the left chest is full of fluid, the left pleural fold is displaced to the right and there is distinct dulness or flatness above, to the right of the midsternum (Fig. 121).

*Auscultation.*—Auscultatory signs in infants and children are most puzzling when the chest is full of fluid, and little diagnostic value can be attached to them in some cases. The chest may be full of fluid while the breathing and the voice may be heard as on

the unaffected side, and pleuritic crepitant râles or crepitations may be heard over the whole chest behind. In other cases, the breathing may be indistinct and distant, and in the lower part of the chest lost entirely. The voice may be bronchophonic in certain localities; it may be of this quality over the whole diseased side of the chest behind, or the tubular sound may be conducted to the healthy side. The voice may be normal above and heard faintly below, toward the base of the lung.

Diagnosis before exploratory puncture rests mainly on (*a*) complete absence of fremitus; (*b*) absolute flatness on percussion with

FIG. 121.



Displacement of the left pleural fold in effusion (empyema) into the left pleural cavity; flatness to the right of the midsternum as indicated. Child, two years of age.

resistance to percussion; (*c*) bronchial voice and breathing over the whole chest behind; (*d*) hyperresonance over the apex, and displacement of viscera, and of the pleural fold in front.

**DISPLACEMENT OF VISCERA.—Liver.**—In infants and young children the presence of fluid may be indicated by displacement of the liver downward on the right side. I have been able to verify the displacement in cases in which large amounts of fluid were present. In infants, the liver is so large and the projection below the border of



the ribs so undetermined, that it is difficult to estimate the exact amount of displacement. The chest is so easily dilated that an ordinary amount of fluid accommodates itself without markedly displacing so heavy an organ as the liver. In children I have been able to make out a displacement of the liver downward before the evacuation of large quantities of fluid. Displacement is of confirmatory value in diagnosis.

*Heart.*—The heart-apex may be displaced toward the median line by fluid in the left pleural cavity. In older children also when the amount of fluid is large the apex is displaced and lies beneath the lower part of the sternum. A small amount of fluid will not always cause displacement, but will find its way around the heart.

**Remarks upon the Diagnosis of Fluid in the Chest, with Exceptional Signs.**—It is not always easy, even for the expert, to decide without puncture as to the presence or absence of fluid in the chest of infants and young children. The following signs will be of service at the bedside.

**DURATION OF ILLNESS.**—If an infant or child has been ill for two weeks or more with signs of pneumonia during the early part of the disease, the physician should be watchful in the presence of the following conditions: If the temperature does not fall, but though remitting still continues; if the signs of consolidation of a small or large area give place to dulness or flatness over a whole side behind, with *bronchophony* over the whole side—for if the condition of the infant is tolerably good, it is evident that such bronchophony may not be due to the total consolidation of the whole lung; if there is displacement of viscera, chiefly of the liver or the heart; if there is drawing inward of the intercostal spaces during inspiration, with real immobility and bulging of a side and dulness or flatness and loss of fremitus.

Fluid is very rarely encapsulated in a small area behind, about the midregion of the chest. Such areas are usually areas of persistent bronchopneumonia. In most cases, there is localized dulness, above and below which there is vesiculotympanic resonance, normal pulmonary resonance or exaggerated resonance. There is distinct respiratory movement of the affected side. On the other hand, a collection of fluid between the lobes of the lungs (interlobar) may give a localized flatness and all the auscultatory signs, such as bronchial voice and breathing, of a local collection of fluid.

*There are certain localities in which the diagnosis of fluid must be made with reserve:*

a. In a case on which I operated, fluid was found posteriorly over the situation of the upper lobe of the right lung. The fluid was completely shut off from the rest of the pleural cavity by a

membrane stretching from the thoracic wall to the interlobar fissure of the lung. Post-mortem showed the case to be tuberculous, the lung on the affected side being the seat of persistent tuberculous bronchopneumonia. I have seen similar cases which were metapneumonic.

*b.* Fluid over the upper lobe in front only, is rare. I have seen one case, but no operation or verification was permitted.

*c.* Fluid over the lower lobe of the lung, in front on the right or left side without corresponding signs behind, is uncommon.

*d.* Circumscribed collections of fluid behind over the middle region of the lung or toward or in the axillary line are exceedingly uncommon.

*e.* In the chapter on the physical signs of pericarditis, it will be shown how a pleurisy or empyema on the left side may be mistaken for pericarditic effusion.

Physical signs having led the physician to suspect fluid, the chest should be explored for two distinct reasons: to determine absolutely the presence of fluid, and to ascertain whether it is serous or purulent.

**Diagnostic Exploratory Puncture of the Chest.**—The INSTRUMENTS necessary are an exploring needle, a millimetre in calibre, and a large barreled aspirating syringe. The needle should not be too short, else it may snap off in the chest. The needle and syringe are boiled for a few moments before being used. The patient is held in the arms of the nurse or mother, so that the posterior aspect of the chest may be exposed. Older children may sit on a table. The chest is scrubbed with soap and water, washed off with ether, then with alcohol, and finally with a solution of sublimate (1:2000). The arms of the infant or child are firmly held and the chest studied in such a manner that should the patient move suddenly the needle will not break in the chest (Plate XVII.).

**INTRODUCTION OF THE NEEDLE.**—The chest is again percussed and the needle introduced into the intercostal space in which percussion elicits the most marked dullness or flatness. This rule should be invariably followed; the needle should not be introduced into any particular intercostal space. On the right side the physician should avoid putting in the needle too low down (liver); on the left side he should avoid introducing it too deeply for fear of wounding a large vessel at the root of the lung. The needle should not be entered too near the vertebral column. The needle having been introduced one or two centimetres, the piston is drawn and held thus a few seconds. Sometimes the fluid is thick and does not flow freely into the syringe. The syringe should not be introduced and then withdrawn and pointed up and down in various directions in quest of fluid, for fear that the struggles of the patient, even if he is firmly



PLATE XVII.



Showing the Method of Making an Exploratory Puncture  
for Fluid in the Pleural Cavity in an Infant.





held, will cause puncture of the lung and bloodvessels. The needle should be withdrawn as rapidly as it was introduced and the whole operation completed in less than a minute. The external wound is covered with a small strip of iodoform gauze held in place with rubber plaster. The needle while in the chest should be held loosely. If it is held firmly, any sudden movement of the patient will cause it to break off in the chest. The needle should not be introduced too deeply for fear that it may enter a dilated bronchus and withdraw purulent secretion which may be mistaken for empyema, or that it may wound the lung and cause hemorrhage.

### Perforating Empyema.

An empyema may perforate externally. In that case there will be an extensive infiltration of the tissues external to the ribs on the affected side, resembling a large phlegmon, and the signs of fluid will persist. If the perforation occurs on the left side, the movements of the heart are likely to be conducted to the external swelling, and there is then what has been called pulsating empyema. The empyema may perforate through the lung, and the signs will then vary with the length of time during which the perforation has existed. It is customary for writers to repeat one another in recounting the physical signs of pneumothorax in a chest in which fluid (pleurisy or empyema) is present. In infants or very young children the following classical signs of pyopneumothorax observed in adults are not commonly found: amphoric breathing, amphoric voice, metallic tinkle, and succussion-sounds. My cases were in children under two years of age. The perforation in the lung must have been too small or too valvular to permit of the entrance of much air into the pleural cavity. These cases at first showed all the signs of the condition which was proved, on introducing the needle, to be empyema. Operation being refused, after a few weeks (three months after the beginning of the disease), the signs changed as follows:

Periodic expectoration of large quantities of pus following coughing spells.

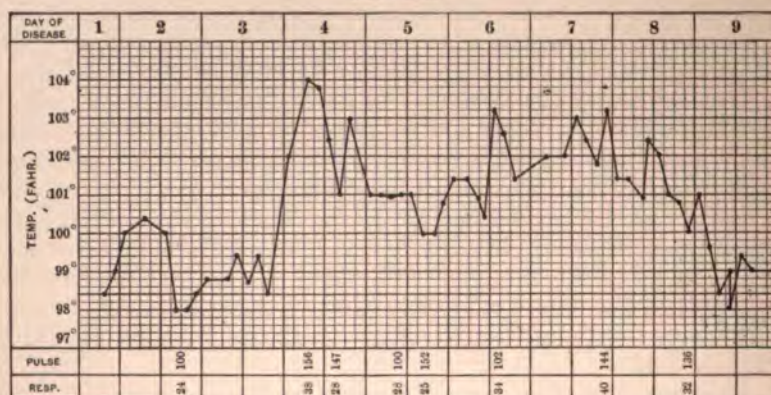
Fremitus diminished over the whole right side and almost lost below.

Dulness over the whole side in front and behind, with tympanic note on deep percussion only. Voice normal; breathing normal—at least not varying from that on the healthy side. In the intervals of expectoration, there were in some cases bronchial voice and breathing.

No succussion-sounds, no tinkling, no amphoric signs. The classical signs seen in adults are met in children above five years of age.

**Course and Termination.**—Pleurisy with effusion and empyema have been considered together, because, in infants and children under two years of age, the effusion in the chest may at first be serous, but subsequently change into purulent exudate. A serous effusion may be followed by a purulent one; it may remain serous and be absorbed as such. Thus it is best, especially in infants, to introduce an exploring-needle into the chest to determine the nature of the fluid as soon as its presence is suspected. In older children also, this may be done at the outset. If a clear fluid is at first obtained and the symptoms do not retrograde within a short time, the needle should be again introduced to determine whether the fluid has remained serous. It is frequently found to be purulent although no infection has occurred as a result of the first puncture. With ordinary cleanliness, the possibility of infecting a serous effusion in the chest and thereby causing it to become purulent is very slight. Purulent effusion appearing after the first exploratory puncture has shown the effusion to be serous, may be due to two causes: either to continuance of the pleuritic inflammation, or to the fact that if the infant or child has lain quietly in bed the purulent elements of the effusion have gravitated to the lower portion of the chest, leaving a clear serum above at the level of the puncture.

FIG. 122.



Empyema, left pleura, followed thirteen days after operation by bronchopneumonia at the apex of the right lung. Male child, twenty months of age. Recovery.

The **prognosis** of pleurisy with effusion and of empyema in infants and children is good. If treated in the proper manner, it is not more serious than the original causal affection. In private practice, the patient being under constant supervision of the physician, the outlook is very good. An effusion can be discovered early and the patient relieved. In hospital practice the results are still good if



the cases are simple and come under treatment before systemic infection has taken place. In my service of 120 cases of all kinds, there were 20 deaths, 4 of which occurred from one to five days after operation. Sepsis had been present before operation and caused the fatal issue. The septic cases therefore give an unfavorable prognosis, as do also those of a tuberculous nature. In the latter, as in other forms of tuberculosis in children, the outlook is better than in the adult and recoveries are not infrequent.

Of the 20 cases of death after operation for empyema, bronchopneumonia either persistent or recurrent caused the fatal issue in 11, general sepsis in 2, marasmus and ulcer of the duodenum in 1, and cerebral embolism in 2. A complicating pericarditis of a suppurative nature may cause death. It is not always possible to diagnose this condition during life. The complication most to be feared in empyema is a bronchopneumonia involving either lung. In many cases the bronchopneumonia is present at the time of operation, or it may come on a week or two afterward during apparent convalescence.

The prognosis of tuberculous empyema is not so unfavorable in children as in the adult. In the former, empyema of a tuberculous nature, like other forms of tuberculosis, may with skilful management make an apparent recovery, though with marked deformities of the chest-wall. In this form of empyema the pleura is thickened, binding down the lung and thus preventing expansion. Extensive rib resections thus become necessary in order to close up the suppurating cavity left by the unexpanded lung.

**Treatment.**—If on exploratory puncture a serous exudate which only partly fills the pleural cavity is found, the expectant plan is followed. The bowels are kept open with an enema or a saline cathartic is administered daily. For this purpose a saline enema, or in older children a teaspoonful of Carlsbad salts in warm water mixed with milk, is efficient. Local vesication is not needed nor is it advisable. The effusion is absorbed if the patients are kept quiet and the diet is easily assimilable. Citrate of potassium in grain  $v$  (0.3) doses every three hours may be given to older children. If the fluid increases in quantity, fills up the chest, causes dyspnoea or pressure symptoms, and is serous in character, the chest should be aspirated. The best form of aspirator for the practitioner is the Potain. The patient is aspirated in the sitting posture. The chest-wall having been cleansed, the needle is introduced in the posterior axillary line toward the lower third of the chest cavity. It is not withdrawn until the flow has ceased or the lung can be felt against the needle in the pleural cavity. As soon as this occurs the needle is withdrawn and the puncture opening covered with a piece of iodoformized gauze. It sometimes happens that there are signs that the chest is filled with fluid and yet very little flows into the

instrument. In such cases the needle should be withdrawn and introduced into the chest-wall at another point. The coughing attack which occurs during aspiration will subside on the patient's taking the recumbent posture. If the chest is quite full of fluid, it is well not to empty it entirely. Sometimes alarming syncope with other signs of cardiac weakness, such as cyanosis, has supervened. If a limited quantity of fluid is removed, the absorption of the rest will follow rapidly.

A daily saline cathartic is given; the patient is kept quiet and allowed a nutritious and easily assimilable diet. The administration of salicylate of sodium may hasten absorption, especially in cases in which there is a rheumatic history. If there is pain or a harassing cough, small doses of codeine should be given.

**Empyema.**—When the presence of pus in the chest is once established, it is imperative that it be evacuated with the least possible delay. In infants and children it is not advisable to temporize by first performing aspiration. Retention of even a limited quantity of purulent exudate in the pleural cavity not only leads to emaciation and physical weakness as a result of continued fever, but general sepsis may also result. Aspiration is not efficient, and is to-day practically abandoned as a mode of treatment. The physician may either incise the intercostal space or resect a rib to obtain drainage.

Simple incision in the intercostal space is efficient in many cases of empyema occurring in the first eighteen months of life. In these frail patients, excision of the rib has been sometimes accompanied by discouraging results.

The greatest number of deaths after any operative procedure for the relief of empyema occur in children under the age of eighteen months. The strength of the patient should be supported as much as possible. A general anæsthetic is not necessary for patients under this age. I find that bronchitis and pneumonia very frequently result from the general use of anæsthetics in young patients. Local anæsthesia is all that is needed. Ethyl chloride in tubes is very efficient. The surface of the chest is carefully cleansed with soap and water, alcohol, ether, and sublimate. An incision two inches long or thereabouts is made obliquely in the tissues over the intercostal space. The space in which a needle has been previously introduced and pus found is chosen. The exploring-needle is always introduced just before operation. Frequently, although pus has been withdrawn from the chest, at a second aspiration none can be found. The theory is that either there was a small localized collection of pus at the first point of aspiration, or that the needle entered a bronchus and withdrew secretion collected there.

On the right side the incision should not be too low, else a tube cannot be retained in the chest on account of the high position of the diaphragm. The seventh or the eighth space in the posterior



axillary line is the best location if pus is present at this point (Fig. 123). On the left side, incisions should not be made too far forward, else the drainage-tube may impinge against the pericardium.

The superficial tissues having been incised, the intercostal muscle is incised, the operator keeping as nearly as possible in the median line of the intercostal space and avoiding the lower border of the upper rib, yet not cutting too close to the lower rib. When the vicinity of the costal pleura is reached, a closed dressing-forceps

FIG. 123.



Empyema, site of incision in line with the angle of the scapula. Infant, twelve months of age.

is introduced into the pleural cavity and opened to widen the puncture. A small drainage-tube or two small tubes are placed in the pleural cavity and prevented from falling into the pleural space by safety-pins passed through them at the distal ends. The pus is not evacuated at the time of operation. The sudden evacuation of fluid which has been retained in the chest for a long time is apt to cause untoward syncopal symptoms. Gibson has made the excellent suggestion that as soon as the pleura is opened the drainage-tube should

be quickly introduced into the chest, the gauze dressings applied, and the pus allowed to escape gradually into the dressings. The dressings consist of a pad of gauze around the tubes, covered by a dry sterilized gauze dressing which is renewed every day. The chest should not be irrigated. No instrument should be introduced into the chest cavity to loosen adhesions. The whole operation is extremely simple, and should not occupy more than a few minutes. Children under five years, and even older ones may be treated by this method. In the older subjects, however, the chest-wall is not so resilient; there are adhesions, and if they are numerous and clots are abundant in the exudate a subsequent excision of the rib may be necessary. On the other hand, the main object of the practitioner in these cases is to evacuate the mass of pus, and incision will

FIG. 124.



Excision of rib for empyema on the right side. Shows the resulting deformity. Five weeks after operation. Child, four years of age.

accomplish this quite as well as the other operation. If subsequently, more drainage is needed, the patient will be stronger and better able to stand the more serious procedure.

Incision is therefore the practitioner's operation even in older children, with whom anaesthesia must however be used. Chloroform is the safest and most easily taken; very little need be



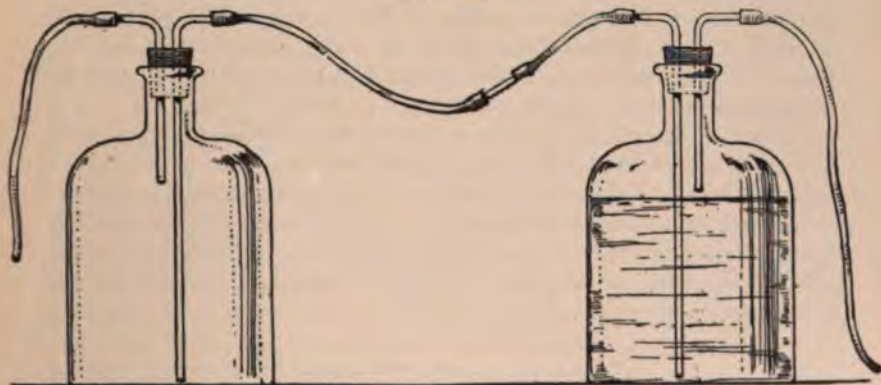
used. As soon as the skin incision has been made, anæsthesia should be suspended.

I perform excision of the rib in all the cases in children above eighteen months, unless there is a contraindication. Severe pneumonia, high fever, cardiac weakness, acute pericarditis or endocarditis, as complications, are contraindications. In such cases incision alone is performed. I excise the rib in the usual way, taking two or three centimetres of rib subperiosteally and incising in the midline of the posterior layer of periosteum to enter the pleural cavity. The finger is not inserted into the pleura to loosen adhesions. After the pleura is opened, double drainage-tubes are introduced by Gibson's method, as in the operation of simple incision.

**Sinus.**—After incision or resection of the rib, a suppurating sinus may remain for months. If a probe introduced into a sinus of this kind impinges against callus or denuded bone, a so-called secondary operation is necessary to take out the denuded rib or callus. This involves a difficult surgical procedure, which it is not necessary to describe here. A sinus of this form will not close until the bone is removed. Temporizing only subjects the patient to the dangers of prolonged suppuration (amyloid degeneration).

**Adhesions binding down the Lung.**—There is another class of cases in which a large amount of fibrin has been thrown out on the

FIG. 125.



James' apparatus for expanding the lungs in empyema.

visceral pulmonary pleura. The lung is thus cramped by an envelope of thickened pleura and cannot expand. A large suppurating cavity or a suppurating sinus is left between the pulmonary and costal pleura. This cavity must be made to close. In such cases the patients are allowed to be up and about. They are taught to blow colored fluids from one bottle to another in the way described by James, of New York (Fig. 125). Two bottles of equal

size, each half filled with the fluid, are used. In simple cases this method is very efficient; in others it is of no avail. The operation of taking out two or more ribs with the intervening pleura must then be performed. In other cases a more extensive operation—the so-called Estlander, in which large pieces of several ribs are excised with the intervening costal pleura—is necessary. If the lung is firmly bound down by a coating of fibrin, the chest-wall must be opened by reflecting a flap of several ribs and the soft parts. The pleura is peeled off the lung according to the method of Delorme. The lung expands, the costal flap is sewn back in its place, and the chest sinus is in time closed as a natural consequence.

The question of irrigating the pleural cavity in the treatment of empyema after operation has been much discussed. As a rule if the temperature drops after operation and remains low, and the discharge is not fetid, no irrigation is indicated. If, however, there are rises of temperature after operation, with a profuse or fetid discharge, the chest should be irrigated once daily with normal salt solution.

**Bilateral Empyema.**—The treatment of bilateral empyema will tax the judgment of the physician. One side, preferably the left in order to relieve the heart, is first operated on by incision or rib exsection; the other side is aspirated, and again aspirated if the fluid or pus accumulates. After a week adhesions will have formed on the operated side, and the strength of the patient will warrant interference on the opposite side. When this is accomplished, the opening on the operated side must be closed by some device, such as a pad of gauze on which is placed rubber tissue covering, and the second side may be operated on by rib exsection or incision.

I have followed this method in two cases without serious accident. The interval of a few days between the operations is sufficient to allow adhesions to form on the operated side to such an extent that, when the second side is opened, the lung of the side first operated on does not collapse. If the sides are operated on simultaneously, the consequent partial collapse of both lungs causes marked symptoms of asphyxia.

### Hemorrhagic Pleurisy.

Simple hemorrhagic pleurisy is not uncommon. It is seen in pleurisy following simple pneumonia, influenza, the exanthemata, and in infants or children in whom there is a tendency to scorbutus. Cases which appear to be rheumatic have been published (Starck). The hemorrhagic form of pleurisy with effusion may occur in very young infants (Lewin, eleven months) or in young children. I have met a number of cases in children who subsequently made a complete recovery, and in whom I could find no tuberculous ten-



dencies. The prognosis in this form of pleurisy is therefore much better in children than in adults. In the latter, a hemorrhagic pleurisy is frequently indicative of a tuberculous factor in the etiology.

### **Hemorrhagic Empyema.**

Hemorrhagic empyema is also not uncommon in infants and children. During the past year I have met four cases in which there was a hemorrhagic exudate. In one case the child was pale, though not emaciated. There may have been a scorbutic element. In another case, in a boy, no such etiology was indicated. In a third case, in a girl, the child was much reduced in health. In three cases the hemorrhagic discharge persisted for days after the chest was opened and streptococci were found in the exudate. In one case the discharging pus was for weeks tinged with blood. In none of the cases were tubercle bacilli found in the pleuritic exudate. Three of the cases made a very good recovery. In these cases also I am inclined to believe that tuberculosis is not always an etiological factor.

### **SUBPHRENIC ABSCESS OR PYOPNEUMOTHORAX SUBPHRENICUS.**

The positive diagnosis of subphrenic abscess should be made with reserve, because no pathognomonic symptom or physical sign of the disease is known. It is a very valuable fact that in 50 per cent. of the cases thus far recorded, the abscesses have contained gas or air. The condition is rare (Maydl) in adults and more so in infants and children. The abscess is situated beneath the diaphragm, and between that organ and the liver. It pushes the diaphragm upward, and may thus encroach on the pleural space and simulate a real pyopneumothorax. A tumor in the lower part of the thorax, which may give tympanitic resonance or tympanitic dullness from the second, third, or fourth rib downward, is thus caused. This resonance may even include the liver, which is displaced downward. Over the region of tympanitic resonance, especially posteriorly, the normal vesicular breathing is absent on expiration and present over the tumor on deep inspiration. It is a peculiarity of the condition that there may be amphoric breathing and metallic tinkle over the tumor, while anteriorly just above it from the second to the fourth rib, there is a sharp transition and normal breathing is heard. Behind, however, on deep inspiration, even over the region of tympanitic resonance, normal breathing may be heard over the lower part of the chest. Over the situation of the abscess the metallic tinkle and succussion-sounds may also be heard. As has been stated, the liver may be displaced downward, crepitations are heard anteriorly over

the liver (perihepatitis), or it may be impossible on account of intestinal conditions to make out the lower border of the liver. I have seen a subphrenic abscess on the left side displace the left lobe of the liver and the spleen downward. The heart is not displaced inward if the abscess is on the left side, but if displaced at all, is so in an upward direction. The lower thorax region may show no abnormalities to inspection, while the upper abdominal region may be normal, painful to pressure, or slightly œdematous.

**Diagnosis and Treatment.**—Exploratory puncture is resorted to in all of these cases. Diagnosis will be aided if the fluid obtained contains, in addition to pus, elements which denote the origin of the abscess, such as food particles, feces, histological débris or pigment from the liver. In many cases the liver suffers from the vicinity of the abscess.

The treatment is surgical.

#### LEADING AUTHORITIES REFERRED TO IN CHAPTER VI.

- Bonnard-Favre*: La température dans la pneumonie. Paris, 1898.  
*Booker*: Johns Hopkins Hospital Report, vi., 1896.  
*Bowditch*: Medical News, 1889.  
*Cestan*: La Thérapie des Empyèmes. Paris, 1898.  
*Czerny and Moser*: Jahrb. f. Kinderheilk., Bd. xxxviii.  
*Finkler, D.*: Die Acuten Lungen Entzündungen, etc., 1891.  
*Fischl, R.*: Volkmann's Vorträge, No. 220.  
*Holt, L. E.*: "Pneumonia in Young Children," Medical Record, 1885.  
*Jürgensen*: Croupöse Pneumonie, 1883.  
*Netter*: Soc. méd. des Hôpit., 1889-91.  
*Neumann*: Jahrb. f. Kinderheilk., Bd. xxx.  
*Pott*: "Pneumonie in Kindesalter," Bib. d. ges. med. Wissen., Bd. iii., 1898.  
*Pratt, I. H.*: "Histology of Acute Lobar Pneumonia," Johns Hopkins Hospital Report, vol. ix.  
*Prudden and Northrup*: "Studies in Pneumonia," etc., American Journal Medical Sciences, 1889.  
*Spiegelberg, I. H.*: "Lungen Entzündung u Magendarmkr.," Archiv f. Kinderheilk., Bd. xxvii.  
*Steffen, A.*: Klinik der Kinderk., etc., to 1895.  
*Stengel, A., and C. Y. White*: "Blood in Infancy and Childhood," Arch. of Ped., April and May, 1901.  
*Terrier and Raymond*: Chirurgie du Cœur, Paris, 1898.



## CHAPTER VII.

### DISEASES OF THE HEART AND PERICARDIUM.

#### HEART.

THE height of the heart and of the great vessels in children does not after the third year materially differ from that in the adult. The ratio of the transverse to the sagittal diameter of the chest in newborn infants is 2 to 1, while in adults it is 3 to 1. This fact should not be forgotten in estimating the size of the heart in infants and children. What in an adult might appear to be a large heart, is normal in an infant or a young child.

**Position.**—In the first year of life the long axis of the heart is more horizontal than in later childhood or in adult life (Rauchfuss). At the third year, the position of the heart is practically that found in the adult (Dwight).

As the child becomes older the heart assumes more nearly the vertical position, and in older children the apex-beat may be found 0.75 to 1 centimetre within the mammillary line. The situation of the mammillary line is variable in young children; the nipple is over the fourth rib, but further removed from the midsternal line than in older children on account of the greater transverse as compared to the longitudinal diameter of the thorax. In older children the heart areas closely resemble those in the adult. In infants and young children there are certain variations from the adult condition which should be borne in mind.

**Size.**—The heart is relatively larger in the infant than in the adult, having 0.89 per cent. of the body weight in the newborn infant, while in the adult it has only 0.52 per cent. (Vierordt).

**Apex-beat.**—The apex-beat in the newborn infant may be felt higher than in the adult. On account of the greater breadth of heart as compared with that of the chest the apex is external to the mammillary line. Steffen says, that normally the apex-beat may be found 1 centimetre external to the mammillary line, or in the mammillary line, or internal to the mammillary line. The apex-beat in infants and children is in the fifth space.

**Inspection.**—Inspection shows in some cases an undulatory movement over the whole cardiac region. This is normal as long as it is confined to the left of the sternum, but an undulatory movement to the right of the sternum is probably indicative of dilatation of

the right ventricle with or without hypertrophy. In rachitis the cardiac region is sometimes unduly prominent. This condition must be distinguished from the more pronounced fulness in the præcordium occurring in cases of hypertrophy or of pericardial effusion. The apex-beat should not be mistaken for an apparent apex-beat which is sometimes seen in young children in whom the intercostal space to the left of the large cardiac dulness is raised with each pulsation of the apex. Percussion in these cases will show the apex to be situated elsewhere to the left and downward. In some cases the apex, instead of pushing the intercostal space forward, draws it distinctly inward. This is in part due to adhesions between the heart, pericardium, and parts external to the pericardium. When children are struggling, the systolic impulse of the heart is seen to be communicated to both the carotid artery and the jugular vein, the vein getting its impulse from its proximity to the artery. The vein may be found to be collapsed and the artery to show an impulse on systole.

**Palpation.**—The following points may be determined by palpation with the tips of the fingers or full palm:

1. Location of the apex-beat.
2. Sometimes the location of the left boundary of the heart.
3. The force of the systole, hypertrophy or dilatation of the heart, especially if pulsation is evident to the right of the sternum.
4. Transposition of the heart to the right.
5. The closure of the valves of the pulmonary artery in the second or third space near the sternum (Steffen).
6. Murmurs which cause friction (pericardial) or thrills (endocardial).
7. Rhythm of the heart action.

**Auscultation.**—In infancy the muscular quality of the first sound is not apparent. The heart-sounds have more the character of the tick-tack of a watch. The muscular character of the first sound fully develops toward the second year of life. All through infancy and childhood there is a natural accentuation of the second pulmonary sound. Too much importance should not be attached to the accentuation even if it is marked.

**Percussion.**—The percussion of the heart has been the subject of much refinement of methods, which only tends to confuse a simple matter. The following method will be found suitable for most clinical purposes:

The lines of demarcation are the midsternal line and a parallel line running through the left nipple. All reckonings as to the limits of cardiac dulness may be safely made from these lines, and such figures will be understood by all physicians. The right border of the sternum is not a good line to reckon from, since the width of the sternum varies. The recumbent posture is preferable in



infants; both the recumbent and upright positions are suitable in older children.

**Method of Locating the Line of Dulness of the Left Ventricle.**—To locate the external boundary of the ventricle, we begin to percuss in the lines parallel with the second, third, fourth, and fifth ribs toward the heart, from the axillary line or the anterior axillary line. To percuss from the midsternal line outward does not in children give as good results.

FIG. 126.



Form of the normal relative cardiac dulness in a child two and one-half years of age.

To locate the external border of the right *ventricle*, we percuss along the *fourth rib* or *fourth space* toward the sternum from the right mammillary line. In young infants a portion of the right auricle and ventricle will be found as high as the junction of the second rib and the sternum (Symington), but it is an ultra-refinement of percussion to try to make out the projection of this part of the right auricle to the right of the sternum. It is found, anatomically, that the curve of the auricle to the right of the sternum begins at the third space, and is most marked behind the fourth costal cartilage. It is sufficient for clinical purposes to make

out this most projecting part of the heart to the right of and behind the sternum.

The apex of the heart is generally made out by percussing along the fifth rib or fifth space from the antero-lateral axillary line toward the midsternal line. The external boundary of the left ventricle is in children slightly outside the apex-beat. The area of cardiac dulness which is absolute and which is uncovered by lung can best be made out by percussing from above downward over the cardiac area. In children or infants this area cannot be marked out as definitely as in the adult. The younger the child or infant, the greater the difficulty. In infants and children interest centres rather in the apparent size of the heart (relative dulness) than in the area uncovered by lung.

The dulness extends to the right and left of the midsternal line, at a level with the fourth rib, as is indicated by the following figures compiled from Steffen's tables :

Infants under one year . . . . .	right v. 4 to 6.5 cm. to right. left v. 3.5 to 6.25 cm. to left.
Children one to two years . . . . .	right v. 4 to 6.5 cm. to right. left v. 4 to 7.25 cm. to left.
Children two to three years . . . . .	right v. 4.5 to 7.5 cm. to right. left v. 4.5 to 6.5 cm. to left.
Children five to six years . . . . .	right v. 5.5 to 7.25 cm. to right. left v. 5 to 8.25 cm. to left.
Children nine to ten years . . . . .	right v. 5.5 to 8.5 cm. to right. left v. 5.5 to 8.5 cm. to left.

Enough has been selected to show that the actual size of the heart as obtained by percussion in infants and children is extremely variable, and that the physician must be guided by the relative size.

**The Pulse.—Rapidly.**—The following table is given by Bednar :

	Beats per minute.
Fœtus . . . . .	108 to 160
First two minutes of life . . . . .	72 to 94
Fourth minute of life . . . . .	140 to 208
Eighth day to second month . . . . .	96 to 130
Second month to twenty-first month . . . . .	96 to 120
Second to fifth year . . . . .	92 to 108
Fifth to eighth year . . . . .	84 to 100
Eighth to twelfth year . . . . .	76 to 96

Respirations in infants, 30 to 32. Pulse-respiration ratio, a to 4. In the second year, 1 to 5 or 6. Turning, crying, and coughing, raise the pulse fifteen to thirty beats per minute. During sleep the pulse falls fifteen to twenty beats. After the third month, the pulse is more rapid in girls than in boys.

**The Rhythm.**—(a) In infants the pulse is normally arrhythmic or irregular both in regard to time intervals and in relation to what is known as the respiration curve.



(b) Dicrotism is a normal characteristic of the pulse in infancy and childhood.

The irregularity of the pulse is in some cases not very marked, in others becomes more apparent under the influence of undue excitement. Dicrotism although very evident and due to the great cardiac elasticity in children (Landois) is never so marked as it is found to be in children who are the subjects of cardiac disease, pertussis (heart strain), or acute infection (typhoid fever).

### CONGENITAL HEART DISEASE.

Congenital heart disease may be suspected from certain physical signs which occur in that condition and are in a sense characteristic of it. These are cyanosis, changes in the area of cardiac dullness, and the presence of characteristic murmurs.

**Cyanosis.**—The cyanosis which is characteristic of congenital heart disease does not occur in any of the acquired cardiac lesions. It is most common in the congenital forms of pulmonary stenosis of the artery, conus, or ostium. On the other hand, it may be absent in marked congenital disease, as in deficient ventricular septum and open ductus arteriosus. In the latter disease it may appear late in the condition, only at intervals, or not at all. It may be absent at birth and appear in infancy or childhood.

**Cardiac Dilatation and Hypertrophy.**—The presence of a murmur of congenital origin does not necessarily indicate change in the area of cardiac dullness. In fact, a normal cardiac area is sometimes evidence of the congenital character of a murmur. Hypertrophy of the left ventricle should be present with hypertrophy of the right ventricle, and a murmur to indicate open ductus arteriosus. Dilatation of the right ventricle is of value when present with a murmur indicating stenosis at the pulmonary valve. On the other hand, marked congenital defects may exist without any change in the size of the ventricle. Moreover, if the cardiac area is enlarged and the apex impulse weak, congenital disease may be suspected. The weak apex impulse indicates dilatation.

**Murmurs.**—The murmur most characteristic of congenital heart disease is a systolic murmur at the situation of the space between the second and third costal cartilage to the left of the sternum, and not conducted into the arteries of the neck. It is only when there are complicated defects that murmurs are conducted into the carotids (open ductus arteriosus).

Fœtal endocarditis affecting the tricuspid or *mitral valves* is rare, and therefore murmurs of congenital origin are rare at these valves.

Diastolic murmurs are, so far as congenital lesions are concerned, of theoretical interest only.

Systolic murmurs, such as those heard in cases of defects of the ventricular septum, and which cannot be attributed to valvular disease, occur at the pulmonic valves. In these cases the murmur has no point of greatest intensity, but is heard not only at the valve, but also over the whole præcordium. The valvular sounds are distinct. The most marked congenital defects or disease of the heart may exist without any murmur or other physical signs during life.

In simple pulmonary stenosis, the second pulmonic sound is weak; in cases complicated with *open ductus arteriosus* and *hypertrophy of the ventricles*, it is accentuated; in cases of pulmonary stenosis and deficient ventricular septum, it is either weak or very low.

**The positive diagnosis of the exact lesion in congenital heart disease** is in many cases impossible. The reason for this is easily found in the fact that if the patient lives longer than the first year, the lesion is rarely simple, but occurs with other congenital defects in the heart. Another cause is the rarity of autopsies on uncomplicated cases which have been carefully studied during life. Lastly, in complex cases, even if the diagnosis has been confirmed at autopsy, it is impossible to say to what degree the lesion diagnosed and the other complicating conditions found at autopsy have been the cause of the signs and symptoms found during life. The physical signs of congenital heart disease vary as the lesion is a simple one or is combined with other congenital defects. The following classification of congenital heart disease of developmental or foetal endocarditic origin will be found useful in clinical work:

1. *Septum Defects*.—Auricular (foramen ovale); ventricular.
2. *Pulmonary Artery*.—Stenosis of the conus, trunk, or ostium: (a) simple cases (before the end of the first year of life); (b) complicated cases with open foramen ovale or ductus arteriosus, defect of the ventricular septum, or transposition of the great vessels.
3. *Aortic Valve Stenosis or General Contraction of the Aortic System*.—The first may be due to developmental defect or to foetal endocarditis; the second, to developmental defect. All aortic conditions anomalous in character have, so far as is known, not been positively diagnosed during childhood.
4. *Valvular anomalies* of the semilunar valves, due to foetal endocarditis or developmental irregularities are of purely scientific interest.
5. *Open Ductus Arteriosus or Botalli*.—(a) Simple; (b) combined with septum defects or pulmonary stenosis.
6. *Transposition of the Heart and Congenital Anomalies of the Pericardium* (of purely scientific interest).

From the above account, which I have modified for practical use



from the classification of Vierordt, it will be seen that only the congenital anomalies of the auricular ventricular septum, the pulmonary artery, and the ductus arteriosus Botalli are of interest to the clinician.

### Stenosis of the Pulmonary Artery, Conus, or Ostium.

This is the most common of all congenital heart lesions. If found after the thirteenth month of life, it is in most cases combined with a congenital deficiency of the septum ventriculorum. Rauchfuss found a simple stenosis in only 10 per cent. of all the published cases. Most of the cases are due to foetal endocarditis.

FIG. 127.



Congenital pulmonary stenosis with open ductus Botalli, as shown by a dull area in the second space above the base of the heart; loud systolic murmur at the pulmonary orifice; clubbed fingers, cyanosis of the general surface, symptoms of chronic bronchitis; dyspnoea on exertion. Boy, twelve years of age.

**Physical Signs.**—Simple stenosis of the artery, conus, or ostium, found only before the thirteenth month (Rokitansky).

**Cyanosis.**—Early and congenital cyanosis with signs of venous stasis, such as clubbed extremities of the fingers, even in young in-

fants. In cases which are met in later life, the clubbing of the extremities of the fingers and cyanosis of the finger-tips are marked.

**Murmur.**—A systolic murmur heard with greatest intensity at the situation of the pulmonary valve to the left of the sternum, between the second and third costal cartilages, and not conducted into the carotids. A weakened second sound at the pulmonary valve; dilatation of the right ventricle.

Simple stenosis is found in infants, but is rare. In most cases there are also present congenital defect of the ventricular septum, open ductus arteriosus, tricuspid changes, or the aorta arises from the right ventricle or both ventricles. The following facts should be kept in mind in the diagnosis of cases occurring after the thirteenth month of life:

If the above signs are present with a weakened second pulmonic sound, there being absolutely no conduction of the murmur into the carotids, it may be assumed that there is a pulmonary stenosis with an open foramen ovale.

Conduction of the murmur into the arteries of the neck, with a very distinct though not accentuated second pulmonic sound, points to the presence of a septum defect with a pulmonary stenosis.

An accentuated second pulmonic sound with conduction of a murmur of a loud buzzing character into the subclavian and carotids, and a hypertrophy of the right and also of the left ventricle, will support the theory of a pulmonary stenosis with a patency of the ductus arteriosus (Fig. 127) (Hochsinger). In these cases of open ductus arteriosus there is a thrill and a distinctly defined area of dulness in the second space to the left of the sternum above the base of the heart. This dulness is of great diagnostic import. It is due to the dilated great vessels at the base of the heart.

As an exception to the above classification, may be mentioned the case of Sansom, in which cyanosis and extreme anæmia were present. In rare cases, the second pulmonary sound may be very low. The murmur may be conducted into the axilla, the right heart not being dilated.

### **Open Ductus Arteriosus or Ductus Botalli.**

This is a very rare congenital defect. There are in the literature only 20 cases of uncomplicated open ductus arteriosus in which the autopsy confirmed the clinical diagnosis. Of these, only 5 occurred in infants under one year of age, and 5 others ranged from the first to the tenth year (Vierordt). The complicated cases occur with stenosis of the pulmonary artery, septum defects of small extent, and open foramen ovale.

**Physical Signs.**—Cyanosis is not present in the majority of cases, or if present is so only intermittently and is not marked.



The **murmur** is a loud buzzing systolic murmur heard with greatest intensity over the pulmonary artery, and not conducted downward, but conducted to the left of the sternum into the veins of the neck (Hochsinger).

There is an accentuated second pulmonic sound which can be heard in the *carotids*.

**Right Ventricle.**—The presence of hypertrophy of the right ventricle tends to confirm the diagnosis; if the left ventricle is also hypertrophied, greater certainty is added. This is of great moment, since hypertrophy of the left ventricle is not present in any of the other congenital defects, except those connected with the anomalies of the aorta and aortic system and which have only a scientific value, since the literature contains no cases which have been diagnosed during life. The dulness in the second space referred to under Pulmonic Stenosis is also of value.

#### **Congenital Defects of the Auricular Ventricular Septum; Defects of Auricular Septum; Open Foramen Ovale.**

Inasmuch as 44 per cent. of the autopsies upon individuals who during life showed absolutely no signs of cardiac disturbances reveal a patency of the foramen ovale, the diagnosis of the condition as an uncomplicated entity should be made with great reserve. This congenital defect is generally found to exist in connection with other defects of a congenital nature (stenosis of the pulmonary artery).

Cyanosis has been found in all the cases in which autopsy has been made. In a case recorded by Foster, there was cyanosis with a varying systolic and presystolic murmur at the sternal end of the third or fourth costal cartilage.

Walshe says that it can hardly be asserted positively that a patency of the foramen ovale may of itself cause a murmur.

#### **Congenital Deficiency of the Ventricular Septum— Maladie de Roger.**

Autopsies have shown that this condition may exist during life without giving any signs of its presence. Moreover, it is so often combined with other congenital heart anomalies, such as stenosis of the pulmonary artery or ostium, that the signs of the ventricular condition must of necessity be obscured by those of the complicating defect.

**Cyanosis** has been present in some cases of uncomplicated ventricular septum defect (Müller) and absent in others. It is present in the cases complicated with pulmonary stenosis.

**Murmur.**—According to Roger, a loud systolic murmur is heard over the whole præcordium, toward the median line, over the upper

third of the cardiac area. According to others (Müller), *the murmur has no special point of greatest intensity*. It is not conducted into the vessels of the neck.

Rauchfuss calls attention to the fact that with this murmur the distinct valvular character of the heart-sounds at the various valves should be heard. The case of Müller was that of a cyanotic infant two months old. A loud murmur having no special point of greatest intensity was heard over the whole cardiac area. The valvular sounds were distinctly heard. Autopsy showed uncomplicated defect of the ventricular septum.

### ACUTE ENDOCARDITIS.

Acute endocarditis is an inflammation of the lining membrane of the heart. That covering the valves and their immediate vicinity is the part generally affected. There is also an inflammation, slight or marked, of the muscle tissue of the heart, and in some cases there is inflammation of the pericardium. Endocarditis thus involves structures of the heart other than the endocardium. Acute endocarditis may be benign or malignant. Between the two extremes, there are all gradations as to severity. All forms of endocarditis are caused by infection which in the malignant variety is of the severest septic type. Foetal endocarditis affects the right side of the heart; after birth, the left heart is chiefly affected. The condition is less frequent before than after the fifth year of life, and occurs with equal frequency among boys and girls (Steffen).

**Etiology.**—Acute endocarditis occurs most frequently with acute articular rheumatism, but may appear in any infectious disease. It is often found in scarlet fever; less often in measles. I have seen it in rare cases of erythema nodosum (2 cases). It may occur with typhoid fever, diphtheria, influenza, pneumonia (Netter), cerebro-spinal meningitis, and tuberculosis. In fact, all forms of sepsis, such as osteomyelitis, either foetal or in the newborn infant or in children, may be accompanied by endocarditis. Endocarditis is present in 16 per cent. of the cases of chorea and is always present in fatal cases of that disease.

**Bacteriology.**—The most important bacteria bearing an etiological relationship to endocarditis are the streptococci of the various varieties and the *Staphylococcus pyogenes*. Harbitz divides endocarditis into the infectious and the non-infectious varieties. He found bacteria in the vegetations in most of the infectious cases, streptococci in 39.5 per cent. and staphylococci in 18.6 per cent. of the cases; other bacteria, such as the pneumococci, were also found. The cases in which no bacteria were found were healed cases. He thinks that the staphylococci most often cause pyæmic endocarditis with ulcera-



tions and metastatic abscess. Welch has, however, found streptococci in ulcerative endocarditis, and does not fully accept the view of Harbitz. The *Diplococcus pneumoniae* is next in importance as an etiological factor. Wright found the *Bacillus diphtheriae* in one case. Other bacteria, such as the *Gonococcus*, the *Bacillus endocarditidis griseus* (Weichselbaum), the *Micrococcus endocarditidis rugatus* and *capsulatus*, the *Diplococcus tenuis* (Klemperer), have been found in cases of adult endocarditis. Although they are all, as well as the *Bacillus typhosus*, doubtless capable of causing the same process in children, actual clinical cases are still to be published.

All forms of endocarditis are thus septic processes due to the circulation in the blood of bacteria or their toxins. In some cases it is possible to discover the point of entrance of the bacteria into the circulation, in others, it cannot be fixed upon. The forms of endocarditis are not so uncommon in infants as is supposed. The tonsil is a great avenue for the entrance of bacteria or toxins into the circulation (Cheadle). It is believed that many cases of endocarditis in children originate in this manner (Packard). I have frequently met with endocarditis in which the only other clinical manifestation was a slight redness or swelling of the tonsils. The integrity of the endothelium of the endocardium must be compromised if bacteria have invaded the tissue of the valvular endocardium (Prudden). It is supposed that the toxins produced by the bacteria circulating in the blood reduce the resistance of the endothelial lining of the endocardium, thus preparing the soil for bacterial invasion.

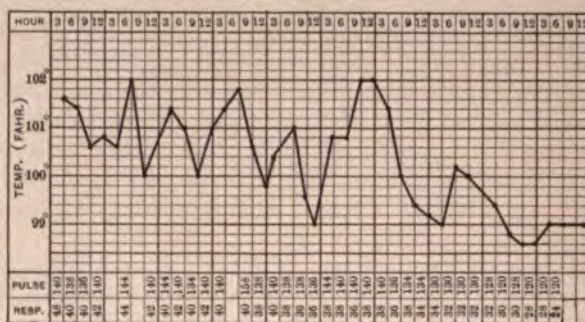
**Morbid Anatomy.**—In some cases the only lesion is a swelling of the valves. They are thickened and succulent, their surface being smooth. The basement substance is swollen and there is an increase of connective-tissue cells (Delafield). In other cases the borders of the valves present transparent, gelatinous, whitish-yellow or reddish formations, varying from the size of a pin's head to that of a bean. These are irregular in shape, cover both surfaces of the valves, and may be single or multiple. They are also seen on the chordae tendineae. The free border of the valve is warty or papillomatous (endocarditis verrucosa or polyposa) (Ziegler). The papillae may appear on the free surface of the valves. There may be a loss of substance with the formation of adherent thrombi of a whitish or reddish color and of tenacious consistency (endocarditis ulcerosa). Small foci of pus may be present in the heart substance (endocarditis pustulosa). Bacterial invasion of the surface of the valves results in loss of substance, formation of thrombi, and changes in the nuclei of the connective tissue (necrobiosis). The mitral valve being more vascular is sooner affected than the aortic or pulmonary valves. Exudation on the valve is replaced by new connective tissue; excrescences and new formations become permanent. If the bacteria penetrate deeply,

thickening of the valve results. Large thrombi are organized, and the valves become shrunken and distorted. Ulceration and loss of substance may result in perforation of the valves. The thrombi just mentioned are sometimes made up of blood-plates; in other cases leucocytes, blood-cells, and fibrin in varying amounts are present.

There may be exudative pericarditis. The myocardium is the seat of degeneration, which leads to dilatation, or to abscess or aneurism of the heart muscle. Through the separation of portions of the thrombi or of the vegetations on the valves, these particles may be carried into the circulation. Containing, as they do, bacteria (mycotic emboli), they cause secondary infections with necrosis or abscess in the kidney, spleen, and brain.

The **symptoms** of acute endocarditis are those of some general infection. They are not in infants and children so characteristic as to direct attention to the heart. Infants cannot and children do not

FIG. 128.



Endocarditis complicating influenza. Second week of the illness. Mitral systolic murmur developed under observation. Female child, four years of age.

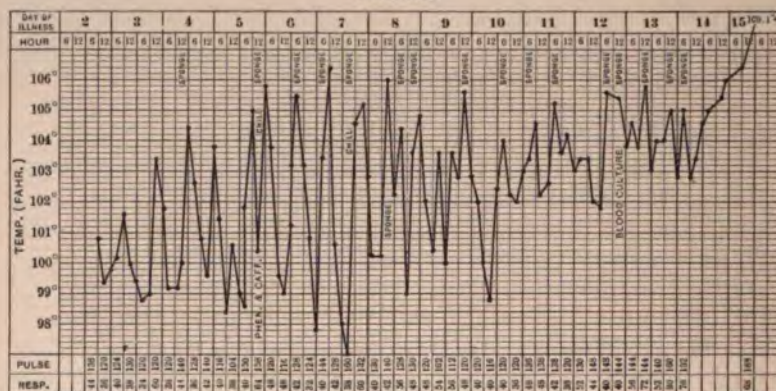
complain of pain, palpitations, or feelings of uneasiness in the heart region as adults sometimes do, and therefore unless the heart is carefully examined as a routine procedure, the simple cases of endocarditis will escape observation. The most interesting cases are those which begin with all the symptoms of an attack of influenza or tonsillitis. There are fever, rapid pulse, and an increase of the respirations to 36 or 40. The fever, however, does not subside in the time occupied by the course of one of the above affections; it continues high, 103°–104°–105° F. (39.4°–40.5° C.), with morning or afternoon remissions. In such cases a most careful examination of the lungs and other organs, fails to reveal anything abnormal. The heart, however, shows the presence of endocardial inflammation. In some obscure cases, there is an increasing pallor with a slight daily rise of a half a degree or a degree in body temperature, which will continue for days or even weeks and give rise to a suspicion of





five years of age or under. The others ranged up to fourteen years. The trend of opinion (Adams) supports the contention of Lazarus, Barlow, and Weichselbaum, that these cases differ from the benign cases only in regard to severity. Dreschfeld divides these cases into the following classes: (a) the primary form, (b) the form complicating septic disease, (c) the form complicating pneumonia and meningitis, (d) the form which occurs as a mixed infection due to septic organisms in the acute infectious fevers or which is secondary to the rheumatic affections of the valves. I have recently observed two cases of septic endocarditis. In one, in a boy with osteomyelitis of the tibia, staphylococci were found in the blood during life. In the other case, which followed a pneumonia, streptococci were found in the blood during life. In the former case hemorrhagic symptoms and signs of severe cardiac disease, such as

FIG. 130.



Fatal septic endocarditis following a pneumonia. Streptococci found by culture in the blood during life. Girl, eight years of age.

gallop-rhythm, were observed. The latter case was seen in my hospital service. The child, a girl of eight years, had had a pneumonia three weeks previous to her admission. She had apparently recovered, had sat up in bed after ten days, and was about. A day before her admission the temperature mounted to 104° F. (40° C.), she vomited, and had diarrhoea. The child showed much prostration, and on examination an area of consolidation was found in the right lung behind. She had an active endocarditis giving a mitral systolic murmur. The liver and spleen were large; the temperature rose and fell twice daily, chills and dyspnoic attacks preceding each rise. The temperature subsided to the normal or subnormal after each rise. There were nausea, vomiting, and signs of cardiac failure. The heart did not at first show any enlarged area of dulness. After a few days the left ventricle showed an increased area of dulness to the



extent of 2 to 3 centimetres outside the nipple-line (acute dilatation), with diffusion of the apex-beat. The right ventricle was dilated. With the extreme fluctuations of temperature, the child became delirious. The heart, as at the time of admission, showed a mitral systolic murmur. After ten days petechiæ appeared, first on the neck and upper thoracic region, and increased both in number and extent. The face and eyes became oedematous (cardiac failure). The patient became unconscious and died in coma with Cheyne-Stokes respiratory phenomena. The blood withdrawn during life showed in culture the presence of long streptococci.

The **diagnosis** of septic endocarditis rests on the history and the presence of cardiac signs, the prostration, the great fluctuations in temperature resembling those in sinus thrombosis in ear disease, the onset of chills and delirium, the presence of petechiæ, and lastly on the results of examination of the blood for bacteria.

Of great interest in this connection, are the cases of chronic recurrent endocarditis which toward the close of the disease have certain symptoms resembling those of the septic or so-called malignant cases. In a child of ten years suffering from chronic recurrent rheumatic endocarditis, there was toward the close of the illness a period during which phlebitis with thrombosis of the deep veins of the neck and arms on both sides and oedema of the corresponding extremities developed successively. After a few weeks the symptoms of phlebitis and thrombosis gradually subsided and there was a period of a few weeks during which the patient was much improved. The fever and anasarca subsided and the heart action was good. Before the fatal issue the endocarditis recurred and there were fever and what appeared to be significant petechiæ on various portions of the body. The case was a rheumatic one and had been under observation for two years. Its outcome gives weight to the theory that a seemingly benign endocarditis may at any time take on a malignant or septic nature.

**Physical Signs of Acute Endocarditis.**—A murmur which develops while a child is under observation is indicative of acute endocarditis.

**Inspection** may reveal nothing abnormal, or there may be extreme irregularity of the action of the heart. There may be increased action, as evinced by visible pulsation over the cardiac area.

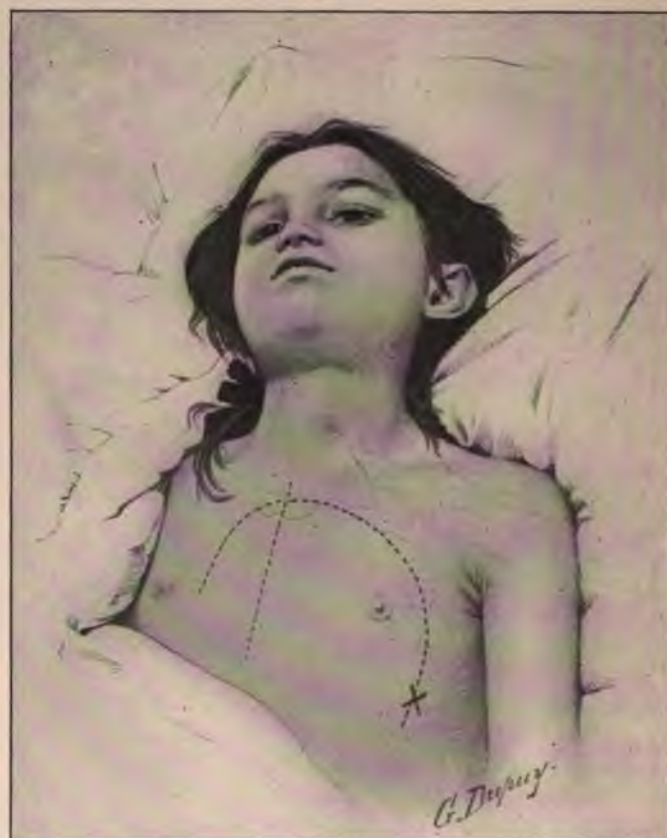
**Palpation** also may reveal nothing abnormal; there may be a thrill over the apex.

**Percussion** at first reveals nothing. In some cases there is a slight dilatation of the left ventricle (Steffen) as the disease progresses. I have seen this dilatation in cases in which the condition had existed for a week. During convalescence the dilatation may retrograde and the heart confines return to their normal limits.

**Auscultation.**—In the majority of cases, a soft systolic murmur

is heard over the apex and the mitral area. There is rarely a pre-systolic murmur. There may be murmurs at the other valves, having the characteristics of the same murmurs in the adult. In any acute disease, the physician should be careful to observe a murmur very carefully before pronouncing it organic. I have found murmurs, especially in typhoid fever in young and older children,

FIG. 131.



Chronic cardiac disease; great cardiac dilatation; recurrent attacks of endocarditis; phlebitis and thrombosis of the deep veins of the neck and arm on both sides successively; œdema of the corresponding arm and forearm; great dilatation of the superficial cervical and thoracic veins. Female, ten years of age.

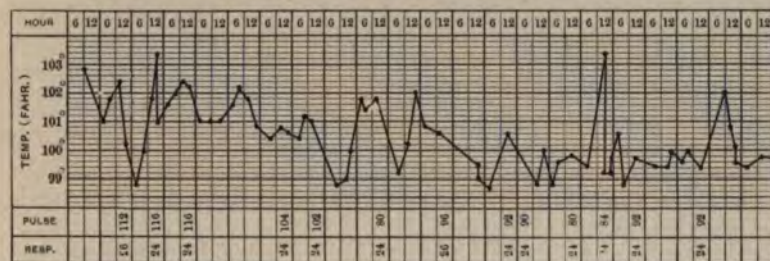
which appeared and disappeared. Such murmurs are hæmic or myocarditic and functional; they are very gentle, generally systolic, and are limited very closely to the apex or pulmonic area. They are not conducted and there are no positive signs of dilatation. Jacobi has met pulmonic murmurs in very young infants, which were at autopsy



shown to be functional. On the other hand, if a murmur is distributed over a valvular area, takes the place of the valvular sound, is conducted into the arteries, and occurs in connection with signs of dilatation, the physician is justified, acute symptoms being in evidence, in assuming the presence of organic disease.

**Course and Prognosis.**—Many cases of endocarditis, especially those not of rheumatic origin, run their course, do not recur, and in after-life give no symptoms referable to the heart. Others run an acute course without developing any physical signs until convalescence. I have seen such forms follow chorea. The murmur develops in the intervals of freedom from symptoms of chorea. Rheumatic cases are likely to recur, and in this tendency lies the danger. The prognosis as to immediate recovery is very good in all of the ordinarily severe cases of acute endocarditis. The severer septic or malignant cases give a grave prognosis. The future of cases of

FIG. 132.



Recurrent endocarditis with acute articular rheumatism which developed under observation.  
Boy, twelve years of age.

acute endocarditis which have recovered will depend very much on the immediate management. I have seen patients who had been allowed to be up and about too early and to participate in sports, develop after a few months symptoms resembling those seen in acute dilatation due to heart strain. These cases show a marked dyspnoea on exertion and cyanosis after play. The children are easily fatigued. They have pain and uneasiness over the region of the heart after running. On percussion an abnormally large heart area is found.

The **treatment** of acute endocarditis is directed toward limiting the damage done by the disease to the heart. Rest in bed is necessary. The patient should not be allowed to maintain the sitting posture, but should be recumbent. The rest should be continued long after the subsidence of the active symptoms. The symptoms and physical signs are the guides as to its duration. If there have been marked disturbance of the heart action and distinct dilatation of the ventricle with signs of myocarditis such as great irregularity of the pulse, the stay in bed should be prolonged for weeks.

If the action of the heart is rapid and tumultuous, an ice-bag should be placed over the cardiac area. This remedy is also useful in cases in which the heart action is not very rapid, but in which there are nevertheless signs of active inflammatory disturbances.

Salicylate of sodium is a favorite remedy, not only in cases with a rheumatic history, but also in septic cases. The dosage is grains  $j$  to  $ij$  (0.6–0.12) every few hours for infants and young children; older children receive more. Some children have stomach pains and disturbances after taking salicylates. There must then in the rheumatic cases be substituted some alkali, such as bicarbonate of sodium. I have administered aspirin in many cases with apparent benefit. A few drops of the tincture of digitalis will be useful in regulating the heart action late in the disease. Digitalis is given for periods of a few days and then suspended for a time, after which it may again be given if necessary. Care should be taken to support but not to drive the heart. The diet should be light, fluid, and easily assimilable. The bowels are best regulated with some saline cathartic or by rectal enemata.

The temperature, if high, may be treated in the same way as in other acute diseases. Baths of low temperature should not be given. The temperature in this disease is of so short duration that in the majority of cases sponging with cold water is effective. The management of choreic cases will be discussed in the section on Chorea.

The injection of antistreptococcic serum in the septic cases has not given satisfactory results.

### CHRONIC HEART DISEASE.

The lesions in chronic valvular disease in infancy and childhood are the same as in the adult subject.

The **etiology** has been considered in the section on Endocarditis.

**Frequency.**—Of 70 of my cases of chronic valvular disease, 37 were of the female and 33 of the male sex; 2 were below the age of two years; 24 from the second to the fifth year, and 39 from the fifth to the tenth year of life. In 50 of the 70 cases the mitral valve was involved, causing either a systolic or a diastolic murmur, or both. The following table will give an idea of the relative frequency of the valvular lesions:

Mitral insufficiency . . . . .	26 cases.
Mitral stenosis . . . . .	6 "
Mitral insufficiency and stenosis . . . . .	18 "
Aortic stenosis . . . . .	6 "
Aortic stenosis and insufficiency . . . . .	1 case.
Endocardial and pericardial disease . . . . .	5 cases.
Combined lesions of mitral and aortic valves . . . . .	8 "

The **physical signs**, the reservations noted in the section on cardiac murmurs being made, are the same as in the adult subject.



On the other hand, certain characteristics of the disease in childhood are not common to the adult subject. There are cases of chronic cardiac disease in infancy and childhood which escape recognition because the heart is not examined with sufficient care. Murmurs of mild intensity pass unrecognized.

There are cases of endocarditis which run an obscure course, give very few symptoms, and which are apt to recur at the onset of tonsillitis or an attack of influenza. These cases of chronic endocardial disease give very few symptoms in the intervals between the attacks. There may be obscure pains in the limbs or joints which are not interpreted by the physician as purely rheumatic, but are believed to be of a grippal character. The patients may eventually develop symptoms of serious cardiac insufficiency. The cases of chronic valvular disease resulting from an attack of some infectious disease may leave the heart little compromised. It is true that upon examination a cardiac murmur which may be marked or slight is heard, but the cases have no subjective symptoms. They

have what is called by the German School a healed endocarditis. They may, however, develop serious cardiac symptoms at the onset of an infection of the gut or other organs. The heart in these cases may be called irritable. The patients do not develop inflammation of the endocardium or pericardium as do the rheumatic cases. On slight disturbance of the gut or intestines, such a heart, even when there is no fever, acts very much like a hypertrophied organ. There is an increase not only of the frequency, but also of the force of the heart's impulse. The vessels are also affected, and there is a bounding full pulse at the radial. As a result of the infection and of the congestion brought about by the increased action of the heart, there will be albumin and casts in the urine. These symptoms subside and do not recur except at long intervals. In the intervals, with the exception of a valvular murmur, there are absolutely no signs of cardiac disease. In children, cases with a slight or marked valvular lesion which are apparently at a standstill, give certain symptoms which are significant of defective cardiac action. On exertion, the children complain of pain in the side or the epigastrium. Examination will show little change in the cardiac areas. The valvular murmur is heard. Such hearts are also irritable. I have often found a distinct history of palpitation occurring at intervals and even in the absence of exertion. Many children with chronic

FIG. 133.



Simple mitral insufficiency; dilatation of the left ventricle. Girl, six years of age.

cardiac disease of a very mild and absolutely quiescent type, exhibit a persistent pallor which does not yield to drugs. Children without other symptoms complain of headaches after slight excitement. Examination will, in these cases also, show a slight hitherto unrecognized chronic cardiac valvulitis. Slight œdema of the eyes which is persistent should direct attention to the heart.

FIG. 134.



Chronic cardiac disease; dilatation of the right and left ventricles. Epigastric pulsation.  
Boy, six years of age.

Many cases without any other signs of chronic cardiac disease show a slight evanescent trace of albumin in the urine.

There may be absolutely no signs of cardiac insufficiency or change in the physical character of the organ. Children with signs of quiescent cardiac disease often have obscure attacks of faintness and vomiting, following every little excitement.

There are also the rheumatic recurrent cases of endocarditis in



childhood. These exhibit very much the same symptoms of cardiac insufficiency as the corresponding cases in adults, viz., enlargement of the liver and spleen. Children appear to recuperate more rapidly than adults, but, on the other hand, the attacks are more likely to recur in them than in older subjects. A compromised heart in a child will bear more strain than in an adult. Cases are frequently seen in which children show on physical examination marked chronic disease, but are notwithstanding exceedingly active and show no symptoms referable to the heart. The signs of insufficiency of the cardiac muscle are the same in children as in the adult. There is dyspnoea on exertion, slight œdema of the general surface, and enlargement of the liver and spleen. In the later stages, there are transudates in the pleura and abdomen. In some cases, especially where there is progressive interstitial myocarditis with adherent pericardium, the pleura may show unilateral transudate.

In cases of cardiac insufficiency, the pulse is persistently high or very irregular. There is persistent dyspnoea. Children with cardiac disease suffer, as a rule, less than adult subjects.

Cardiac angina is not an uncommon symptom in cases of aortic disease. It is present in cases in which there are signs of lack of compensation. The angina comes on in attacks occurring chiefly at night, and is very severe. I have seen a boy of eight years with an aortic murmur suffer from these attacks for days. In such cases there are a dilated ventricle and an enlargement of the liver and spleen.

The **prognosis** of chronic valvular disease in childhood depends very much on the type of disease. If the heart is only slightly affected and the patient not a rheumatic subject, the outlook is good. With careful management all ill after-effects can be avoided; children thus affected may grow to adult life without suffering from any symptoms referable to the heart. If, on the other hand, they are attacked by any intercurrent disease, such as scarlet fever, the heart may again become the seat of inflammatory processes. The patients may, however, recover and continue free from symptoms for years. The rheumatic cases give the most unfavorable prognosis. These are prone to recurrent attacks of endocarditis, each attack leaving the heart in a more weakened condition than before. Most of my cases have been in children who, having had one attack of rheumatic endocarditis, suffered from the affection to a greater or lesser degree for years. Within a few years of the first attack they succumb to progressive non-compensatory cardiac disease.

**Treatment.**—Many cases of cardiac disease in infancy and childhood give no symptoms and need very little treatment beyond careful and judicious management. Children thus affected should have a carefully regulated dietary, and should not indulge in sports which subject the heart to strain. They should not ride the bicycle, but may, however, indulge in many of the amusements of children,

such as skating, roller skating, swimming to a moderate degree, and horseback exercise. They should be under constant observation, and when attacked by any acute infection however slight should be put to bed, and kept quiet until long after convalescence. In these cases an antirheumatic course is pursued even although the illness be only a mild attack of influenza or tonsillitis. It is well to give the salicylates in small doses for several days and to keep the bowels open with some alkaline cathartic. With children who suffer from rheumatism, the nature of the primary disease should not be forgotten. They should have constant antirheumatic treatment even when the cardiac disease is at a standstill. Heiman has recently shown the beneficial effects of the intermittent administration of salicylates in these cases. I have carried out that method for some time. Alkaline baths of the Nauheim form are of great utility in alleviating the subacute rheumatic pains from which these subjects suffer, and this treatment also tends to keep the rheumatic tendency in abeyance. Such children should be kept under constant observation. The temperature should be taken twice daily. Any rise of temperature should be regarded as a threatening sign and the patients put to bed for perfect rest until the crisis has passed. In cases in which there is marked dilatation or pericardial involvement, any exacerbation of symptoms is a signal for immediate rest in bed. Slight œdema of the surface and swelling of the liver and spleen will subside if treated with perfect rest, a light assimilable diet (milk), and mild alkaline catharsis. It is not always necessary to use digitalis. If given at all, it is best administered in the form of the infusion. I am accustomed to use this drug for a period of two or three days, after which I discontinue it. There is no doubt that its action continues after the administration is stopped. Convallaria in the form of the fluid extract is at times one of the most useful remedies in cases in which digitalis has failed to give relief. If there is great dyspnoea or orthopnoea, codeine in moderate doses should be used. Young children do not bear morphine well. It certainly should not be used hypodermatically. In aortic disease in older children, nitroglycerin in doses of grain  $\frac{1}{100}$  (0.0006) relieves the angina. I administer morphine only to older children, and then only when the nocturnal attacks of angina are very severe. I have not found strychnine very useful in the chronic forms of cardiac disease. Caffeine in moderate dosage seems more useful in correcting the irregularity of the pulse or bradycardia seen in some of these cases. In combination with digitalis it gives excellent results. If ascites appears, the patient should be promptly tapped to relieve the circulation and the abdomen supported by a binder. If there is a pleuritic effusion at the same time, it should not be disturbed. With relief of the abdominal distention, the pleuritic effusion often disappears.



### CARDIAC MURMURS.

Cardiac murmurs which are the result of disease or insufficiency of the valves of the heart have the same general character as those in adults, the following being the chief points of difference :

*a.* Cardiac disease of a very serious character may exist (as in congenital cyanosis) without any murmur.

*b.* Cardiac murmurs are as a rule louder in children than in adults. The loudness is therefore no guide as to the seriousness of the affection.

*c.* Cardiac murmurs in children are sometimes heard loudly conducted over the whole chest ; diagnosis of disease of a particular valve must be based on the greatest intensity of the murmur at that point.

*d.* Hæmic and dynamic murmurs in children under four years of age are not so common as is supposed. There should be no hesitation in making the diagnosis of organic affections in systolic, basic, or apex murmurs if there are distinct conduction or signs of dilatation or hypertrophy. This is especially to be remembered in chorea, extreme anæmia, and in febrile affections where rapidity in time and rhythm (galloprhythms) causes adventitious sounds.

*e.* The conduction of the aortic murmurs into the arteria femoralis occurs in occasional cases in children. Pulsation of the liver or spleen, as found in aortic disease of adults, is not present in children (Steffen).

#### Accidental Cardiac Murmurs in Infancy and Childhood.

Accidental murmurs are divided into those heard over the heart, in the arteries, and in the veins. The study of the accidental murmur of the heart in infancy and childhood has been much neglected. West and Hochsinger give the most valuable data. The principal points of difference between the murmurs in infants and children and those in the adult are as follows :

**Cardiac Murmurs.**—*Anæmia.*—The severest forms of anæmia sometimes fail to give hæmic murmurs. Not one of 200 cases under four years of age examined by Hochsinger gave anæmic murmurs. After the fourth year and up to the seventh year of life the frequency of the anæmic and hæmic murmurs increases. I have in very exceptional pernicious anæmias found a mild blowing basic murmur. One such case occurred in a child under four years.

*Fevers.*—The hæmic murmurs so common in the febrile affections of adult life are rarely heard even in severe febrile affections with anæmia, in patients under the age of three years. I have heard them in children under three years of age, with severe typhoid fever. They are common in typhoid fever in older children.

*Characteristics of Anæmia Murmurs.*—These never occur with signs of cardiac dilatation or hypertrophy. They are not conducted into the arteries. They never entirely take the place of the valvular cardiac sounds, but accompany them. They are soft blowing murmurs, heard at times most loudly at the pulmonary valve, sometimes heard over the base and whole præcordium, and faintly heard at the apex. They are never heard at the aortic or tricuspid valves, or behind. They are inconstant, disappearing for a time and again appearing at the various points of the chest.

**Accidental Arterial Murmurs.**—The theory held by some observers, that pressure of the stethoscope on the arteries of the neck may cause a murmur, should be entertained with caution. Correct stethoscopy will hardly lead to such an error. A murmur in the large arteries of the neck is conducted from the heart and is invariably organic in origin. I have heard aortic murmurs conducted in the femoral artery.

**Venous Hum.**—Although cardiac accidental murmurs due to anæmia are rarely heard in children, the venous hum due to the same cause is frequently heard. In young infants and children it is present in the veins of the neck, is quite loud, and is heard at either side of the upper part of the sternum. If there is anæmia due to valvular cardiac disease, the venous hum is heard in the arteries of the neck, with the organic murmur.

### MYOCARDITIS.

Myocarditis is very frequent in infancy and childhood. Most of the knowledge of this condition has been obtained from a study of the disease in young subjects. This is due to the fact that in early life the heart is especially exposed to the deleterious action of the toxins of the infectious diseases. Myocarditis is a degeneration or inflammation of the muscular substance of the heart, secondary to the action of poisons (phosphorus) to the toxins of bacteria (as in the exanthemata, typhoid fever, diphtheria, pertussis, sepsis, osteomyelitis), or to the changes consequent upon disease of the pericardium, or endocardium, of rheumatic or infectious origin.

**Morbid Anatomy.**—If there is degeneration of the myocardium, only the muscular fibre may be the seat of fatty changes. There is an increase of fat drops in the muscular tissue of the heart. In advanced conditions, the fatty changes are apparent to the naked eye as a yellowish discoloration beneath the endocardium. In other cases, there is a granular or hyaline degeneration of the muscle fibre or a vacuole formation. The cell protoplasm becomes cloudy, hyaline, loses its striation, and disintegrates or is replaced by drops of fluid. This occurs in diphtheria, typhoid fever, pneumonia, chronic con-



gestion, and in toxæmia of various kinds. Thrombi may form in hearts which are the seat of advanced degeneration. In toxæmia and the infectious diseases, there is inflammation of the myocardium. There is an invasion of the muscle tissue by bacteria from the endocardium (staphylococci, streptococci, and pneumococci). In such cases, there are also grayish or yellowish discoloration of the muscle tissue, vacuolization, and granular and hyaline degeneration. The muscle tissue is the seat of small cell infiltration or there may be abscesses of microscopic or macroscopic size. If recovery occurs these areas may cicatrize with connective tissue. Tuberculous and syphilitic inflammations of the myocardium occur, but are uncommon.

**Etiology.**—The degenerative or inflammatory changes may be caused by the direct action of the bacteria (Almquist), but usually the influence of the bacteria themselves is only slight, since they do not find in the myocardium a favorable soil for growth. The toxins of these bacteria produced either elsewhere in the economy and circulating in the blood, or in the heart muscle itself, are chiefly instrumental in causing the degenerative changes (Welch, Flexner, Schamshin). Fever, as such, has only a slight influence in causing myocarditis (Werhofsky).

The **symptoms** of myocarditis can best be understood by studying the heart in the various infectious diseases. In diphtheria, myocarditis may be suspected if there occur sudden syncope, faintness, chilly sensations, vertigo, and vomiting. The patients complain of præcordial weakness; there are all the symptoms of collapse and a flickering, irregular pulse. These phenomena may appear at intervals throughout the disease and persist far into convalescence.

In acute forms of pneumonia in which the toxæmia is very great, infants may, even at the outset, exhibit cardiac weakness. There are slight cyanosis of the lips and abnormal pallor of the face and general surface. The heart action is more rapid than in other cases of pneumonia in which the lung lesion is quite as extensive. At the crisis, the action of the poison on the heart is evinced by an irregularity or arrhythmia of the pulse. The pulse may be extremely slow (bradycardia). In septic conditions there will, late in the disease, be galloprhythm, distortion of the pulse-respiration ratio, cyanosis, and extreme præcordial distress. Hensch, Osler, and I have shown that there may be degenerative changes in pertussis. These are clinically apparent in cases which have extended over a long period. A constant dyspnoea, an abnormally high pulse-rate, drowsiness, disinclination to exertion, and slight œdema of the face and other parts of the body are present. In rare cases physical examination reveals a slight dilatation of the right ventricle. In other cases there is at the apex a faint systolic murmur of purely muscular origin. In

adherent pericardium, the advance of the process into the myocardium is indicated by the symptoms above detailed.

The myocarditis of chronic valvular disease is a progressive process. It manifests itself by the signs of lack of compensation described in the section on Chronic Cardiac Disease. The varying pulse, the dyspnoea, the enlargement of the liver and spleen, and transudates into the serous cavities, all indicate this form of progressive weakness of the cardiac muscle.

**Diagnosis.**—Although the diagnosis cannot in all cases be made with absolute certainty, the presence of the condition may be suspected if the following sets of symptoms appear at regular intervals in the course of the disease—attacks of palpitation and faintness, pallor, cardiac irregularity, gallop rhythm and weakness of the apex beat and of the first muscular sound of the heart, with intensification of the second pulmonic sound.

The **treatment** should support the heart and lessen its work, and should also be directed toward the management of the primary condition. In all of these cases, prolonged rest for the heart, continued long after convalescence, is of primary importance. It should not be forgotten that even in a degenerated organ there is healthy tissue on which the drugs and treatment act. These healthy foci should be sustained, and not exhausted by the action of powerful drugs given in large doses. Degeneration cannot be cured by drugs; nature must heal the diseased areas.

### PERICARDITIS.

Pericarditis is an inflammation of the pericardium due to infection, which may take place through the blood- or lymph-channels or may occur through contiguity to infected areas in neighboring structures. The existence of primary pericarditis or so-called idiopathic pericarditis apart from rheumatism or infection is a matter of doubt. It is therefore to be regarded as secondary to other conditions or the result of direct systemic infection.

**Occurrence.**—Pericarditis occurs in foetal life (Billard, Tardieu, Heiter); Bednar describes cases in newly born infants; it is common in infancy and childhood. Steffen and Baginsky describe a number of cases occurring in infancy. Of 66 cases of pericarditis in children, Baginsky found 20 to occur during the first year of life. The next greatest frequency was between the first and the fifth year.

**Etiology.**—The majority of cases occur as complications of acute articular rheumatism (Steffen, Friedreich, Bauer, Baginsky), with or without chorea. Tuberculosis and pleuropneumonia rank next as etiological factors. Pericarditis occurs in the exanthemata, scarlet fever, measles, and typhoid fever. It may complicate pertussis,



diarrhoeal disorders, otitis, meningitis, peritonitis, mediastinitis, or any septic process, such as osteomyelitis. It is also in the newly born infant concomitant with septic conditions. Finally, traumatism may cause pericarditis. The tuberculous form is uncommon before the fifth year of life (Sée).

**Bacteriology.**—The pyogenic bacteria most frequently found in pericardial effusions, and which play an etiological rôle, are the pyogenic streptococci and staphylococci, the pneumococcus of Fränkel and Weichselbaum, the tubercle bacillus, the Friedländer bacillus, the *Bacterium coli*, and the *Bacillus pyocyaneus* (Ernst).

**Forms.**—There are the same forms of pericarditis in children as in the adult subject. The forms with effusions have, however, a tendency to become purulent, especially in infants and younger children (Baginsky). In these patients, the fibrinous forms result in localized or general adhesions of the two layers of the pericardium and in partial or complete obliteration of the pericardial sac (adherent pericardium).

**Morbid Anatomy.**—In the mildest forms, there is only a loss of lustre to the serosa in circumscribed or diffuse areas. The fluid in the pericardial sac may be increased in quantity and may contain cellular elements. In other forms, the surface of the pericardium is coated with a layer of fibrin of greater or less thickness. The fibrin may be in the form of bands or of small villous formations. There may be minute hemorrhages on the surface (Delafield). In more pronounced processes the fibrin is in the form of hemorrhagic tenacious masses forming a thick network of strips or bands (cor villosum). The quantity of fluid in the sac varies. The fluid may contain blood.

In the first stage of inflammation, the connective tissue of the pericardium is infiltrated with lymphoid cells and the vessels are filled with blood. After the third day, new vessels appear in the fibrinous exudate on the surface. Fibroblasts, spindle-shaped, spherical, and branching, form a network in this new tissue (Ziegler). Granulation tissue and finally new connective tissue replace the fibrinous exudate, after a period of weeks (productive pericarditis). The so-called opaque areas of thickened pericardium, the maculæ tendineæ seen in adults, are rare in children (Steffen). Adhesions, either localized or general, may form between the two layers of the pericardial sac, causing its partial or complete obliteration.

**Tuberculous forms** of pericarditis may occur as miliary infiltration of the parietal and visceral layers of the pericardium. There may be serous, serofibrinous, purulent, or hemorrhagic exudate in the sac, or gray cheesy nodules of tubercle tissue may be present in the epicardial and subpericardial tissue (Ziegler, Baginsky).

**Myocarditis**, circumscribed or general, may occur in all forms of pericarditis. The adhesive forms are complicated with myocarditis.

**Symptoms.**—Pericarditis in children manifests itself by rational symptoms and physical signs.

**Rational Symptoms.**—At the bedside, the symptoms of the different forms of pericarditis cannot be divided into classes. Some of the fibrinous or dry forms run an insidious course without giving any marked symptoms of the disease. On the other hand, large effusions may make their appearance without any previous rational symptoms which are characteristic. This is the case in the forms of pericarditis in infants and children, which occur in septic conditions, in pneumonia, empyema, and in the exanthemata. On the other hand, if attention has been drawn to the heart, it will be found that certain symptoms may be traced to the inflammatory process in the pericardium. If the patients have been suffering from endocarditis of rheumatic origin, empyema, or one of the exanthemata, they show the symptoms of grave cardiac disease. They have an anxious facial expression, with marked pallor and cyanosis of the lips. They do not, as a rule, complain of pain. The respirations are markedly increased, as is also the pulse. Older children may complain of pain or uneasiness in the epigastrium. They also show marked dyspnoea and orthopnoea. In infants there are signs of pain on breathing. In some of the fibrinous forms there is fever, but dry forms of pericarditis may run their entire course without it. The purulent forms give a remittent temperature-curve. The pulse is rapid, varying from 120 to 150. In the forms with effusion, the pulse is irregular. If myocarditis is present, the pulse is irregular and persistently high, and there is an accompanying increase in the number of respirations. There is no case on record in which the diagnosis of mediastinopericarditis has been made in a child during life and confirmed at autopsy, nor does the so-called pulsus paradoxus give any assistance, since it is present in other conditions in childhood (Steffen).

**Physical Signs.**—In pericarditis, there are the physical signs of the dry plastic forms and the forms with effusion into the sac. The signs of the dry pericarditis and those of the first stage of that with effusion are practically identical and may be considered together.

**Inspection.**—In dry plastic pericarditis and the first stage of pericarditis with effusion there may be no signs to be detected by inspection. On the other hand, there may be an increased action, apparent to the eye, over the whole cardiac area to the left. When effusion takes place, little or no pulsation can be made out over the cardiac area when the patient is in the recumbent position. There may be distinct bulging of the cardiac area, varying with the amount of fluid present. No localized apex impulse is visible when the amounts of fluid are large. There may instead be a diffuse pulsation over the area of the apex and toward the sternum.



**Palpation.**—In dry pericarditis, and in the first stage of pericarditis with effusion, there is a friction fremitus felt over the areas in which the friction murmur is heard. This may be at the apex, at the base, or along the right ventricle close to the left border of the sternum.

**The Apex-beat or Impulse, and its Relations to the Chest Wall in Pericarditis with Effusion.**—As effusion takes place, it is indicated by certain physical signs relative to the heart apex, and by the line of dulness to the left. Investigations have shown that, when the patient is in the recumbent posture, pericardial effusion first collects at the base of the heart around the great vessels. It next collects over the anterior surface and in the anterior-inferior cul-de-sac of the pericardium (Voinitch). When the patient is *recumbent* the effusion does not necessarily push up the apex-beat. On the contrary, it separates the heart from the anterior chest wall. In moderate effusion the apex-beat may still be felt in the normal position. As the effusion increases, the apex-beat recedes and becomes less discernible and more diffuse, and in large effusion may disappear. This is especially the case, if there is dilatation of the heart or adhesions at the apex. When the effusion is again absorbed, the apex-beat becomes evident in the former situation.

When the patient is *sitting*, the pericardial effusion collects beneath and behind the heart, and, if the heart is not enlarged or held down by adhesions, the apex-beat may at first be *displaced upward*, and will be felt above and to the outside of its normal position. These facts will explain the failure in certain cases of pericarditis, to obtain the displacement of the apex-beat upward. In one of my cases, a boy of six years, suffering from chorea, endocarditis, dilated heart, and pericarditis, the apex-beat was observed in the beginning of the stage of effusion to be located in the sixth space, slightly outside the nipple line. Effusion having occurred, the apex-beat could still be observed in its former locality, but the area of absolute dulness indicating effusion extended beyond the apex, four cubic centimetres to the left of the mammillary line. The effusion disappeared and the apex then corresponded with the line of dulness of the left ventricle.

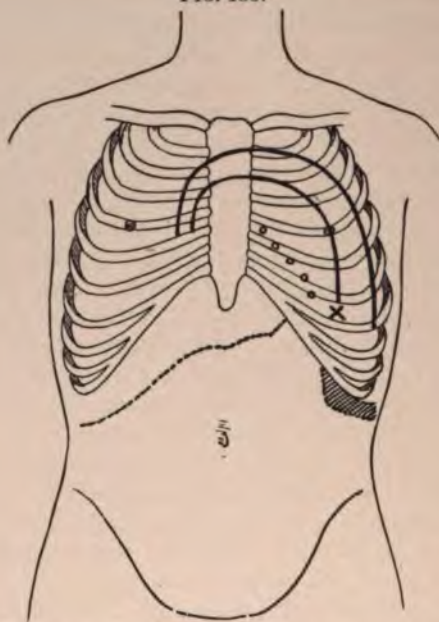
**Percussion.**—In dry fibrinous pericarditis, and in the dry stage of pericarditis with effusion, there is no increase in the area of cardiac dulness directly traceable to the disease. If there is a slight dilatation or relaxation of the ventricle due to myocarditic complication, the normal præcordial dulness may be more distinct.

The effusion must have a bulk of 40–60 grammes ( $1\frac{1}{2}$  to 2 fluid-ounces) before definite signs of its presence can be obtained.

In children, the area of dulness due to pericardial effusion does not have the triangular shape seen in adults. The position of the heart is more horizontal and its shape is retained by the distended sac.

Thus, to the left, the dulness may extend in a curved line outside the situation of the nipple. Superiorly, it may extend as high as the first rib. It then extends in an almost horizontal line two or more centimetres to the right of the sternum (Fig. 135). The line of dulness

FIG. 135.



Pericardial area of dulness due to effusion in boy, six years of age. Chorea, endocarditis, and pericarditis: x, apex-beat before effusion; o o o o, friction murmur; outer curved line shows general shape of distended pericardial sac.

to the right of the sternum then extends downward in an almost vertical line to the liver (sixth space) (Steffen, Baginsky, Ausset). These facts are very important in differentiating dulness resulting from pericardial effusion from dulness due to other causes. Even in moderate effusion there is resistance to the percussing finger. If the patient's position is changed from the recumbent to the sitting posture, the heart falls forward, the pericardial sac is distended, and the dulness to the left may come more toward the mammillary line and, to the right, toward the sternum (Baginsky).

**Auscultation.**—The friction sound is diagnostic in dry plastic pericarditis and in the first stage of pericarditis with effusion. It may, at the outset, be heard at the apex (Steffen), but is also heard to the left of the sternum over the base, or below, to the left of the sternum, over the fourth or fifth space. Steffen finds it in children, at first, most frequently at the apex. The murmur may be heard on



systole or diastole, or on systole only. It may or may not accompany the valvular sounds. It is of very limited distribution, is not conducted, and is of a fine crepitant quality or has a rubbing or a rasping or clicking sound. In the case of a boy suffering from recurrent chorea and pericarditis, there was a loud scraping friction at the apex with murmurs of mitral and aortic regurgitation. I was able in this case to confirm the statement of Walsh, that a loud pericardial friction may sometimes be heard behind, between the scapulæ, to the left of the spine. The friction may for the first day or two be of a crepitant quality and then acquire a rubbing quality. I observed this change in a child four years of age. The patient suffered from dilatation of the left ventricle with mitral insufficiency and stenosis with pericarditis. The friction for two days was crepitant in quality and just audible over the fourth and fifth spaces, to the left of the left border of the sternum. After two days, the murmur of friction acquired a loud rubbing quality. The murmur is sometimes very evanescent or may disappear or reappear at short intervals. The sounds may be intensified by causing the patient to lean forward. When effusion appears, the friction sounds may entirely disappear, or may be heard only in areas around the great vessels or indistinctly over the præcordium. A knowledge of these facts is important in making a diagnosis of fluid in the pericardial sac. The friction sounds may reappear on absorption of fluid. Pleuropericardial friction sounds are rough or fine sounds obtained in children as in adults with the respiratory movements of the lung. They are intensified on expiration and disappear when respiration is momentarily suspended. They may be heard over any part of the præcordium. They are caused by the rubbing of the inflamed pleura and pericardium against each other. This friction is limited to one edge of the cardiac area, generally the left, and is sometimes heard in the back, on the left side.

The **diagnosis** of pericarditis can only be made from the physical signs. In dry plastic pericarditis and the first stages of pericarditis with effusion, the friction sound is the diagnostic sign. If a pericardial friction is once obtained, careful watch should be kept for the appearance of fluid. It is not possible at the outset to differentiate a dry pericarditis which will remain as such, from the first stage of a pericarditis with effusion.

In the stage of effusion, small amounts of fluid will sometimes escape diagnosis. This is likely to occur if a process such as empyema is in progress on the left side. The first stage of a pericarditis may escape diagnosis if the friction sound is evanescent. If the effusion appears in considerable quantity over the great vessels, percussion is made in this region, especially to the right side of the sternum at the level of the second or third space, for an increase in

dulness due to a distended pericardium. Absence of dulness in this region across the sternum and for a few centimetres to the right of the right border is presumptive evidence against the presence of any considerable effusion. If dulness exists to the right of the sternum, low down only on a level of the fourth interspace, there is probably no pericardial effusion, but, instead, dilatation of the right ventricle.

**Differential Localization by Percussion of Pleural and Pericardial Effusions.**—In cases in which pericardial effusion is very large or in which there is pleural effusion into the left side of the chest, a question may arise as to whether there is a simple pleural effusion general or localized, pericardial effusion, or both. Percussion along the sternum will in simple left pleural effusion easily mark out the displaced left pleural fold. Even if there are large amounts of fluid, the fold of the left pleura will be found to be distinctly displaced toward the right border of the sternum. The pleural line will never, except under very exceptional conditions, pass beyond the border of the sternum to the right. If large pericardial effusion is present, the dull note of the effusion extends beyond the right border of the sternum. In left pleuritic effusion the apex of the heart is found by auscultation to be distinctly displaced to a situation beneath the sternum, while in pericarditis it will at first be found to be in the normal position and subsequently to disappear or to be displaced upward and outward.

The **prognosis** of rheumatic pericarditis is good. The purulent forms of pericarditis are in the great majority of cases fatal, especially in very young infants. In older children, I have seen cases of purulent pericarditis, due to infection from a concurrent pneumonia or empyema, recover with timely pericardotomy. The septic forms of purulent pericarditis, complicating sepsis of the newly born and forms of osteomyelitis, are fatal.

The **treatment** of the dry fibrinous forms of pericarditis is limited to the relief of the pain and the treatment of the primary condition, rheumatism. The pain is best relieved by the administration of mild opiates. Codeine in small doses is efficient in many cases. I am not in favor of blistering the præcordial region in children, or of applying a seton, as is done in adults. If the heart is tumultuous, small doses of digitalis in the tincture form and the constant application of an ice-bag over the præcordial region are the most effective remedies. Some authors believe that the ice-bag is also a very powerful means of limiting the inflammation. In rheumatic or choreic cases the salicylate of sodium is given, or if this disagrees with the patient, the ordinary bicarbonate of sodium in doses of grains  $\times$  (6.5) three or four times daily. Perfect rest in bed, long after the process has run its course, is indicated, on account of the ill effects of strain on the heart after the myocarditic changes which are undoubtedly present in many of the cases.



When effusion has taken place, the question of the advisability of puncturing and exploring the pericardium always arises. It is very difficult to choose the proper time for entering the pericardium. I have had a number of cases of pericarditis with effusion recover without being subjected to what is at best a hazardous procedure. I can only detail my own practice in these cases. I temporize until the orthopnoea and cyanosis are extreme and evidences of pressure are marked. Too much importance should not be attached to ordinary symptoms. On the other hand, if the temperature is high and daily remits to near the normal, there may be a purulent effusion. If after a reasonable length of time the patient steadily loses ground and the signs of effusion are marked, the pericardium should be entered to determine the character of the exudate. If it is serous, ordinary aspiration will suffice, but if purulent, the operation of pericardotomy should be performed. Pericardial puncture or incision is performed in the same manner as in adults.

It may be remarked that Henoch has never punctured the pericardium. In one of his cases, post-mortem examination showed small sacculated purulent collections of fluid which could hardly have been evacuated by a single puncture. I found a similar condition post mortem in a case in which puncture of the pericardium was undertaken, and resulted in puncture of the heart.

### ADHERENT PERICARDIUM.

Adherent pericardium is an agglutination, localized or complete, of the visceral and parietal walls of the pericardial sac which becomes partly or completely obliterated. The condition follows either a dry plastic pericarditis or a pericarditis with effusion, in the stage of absorption. In the latter case, if the absorption of fluid has been observed and the redux friction-sound obtained, adhesion of the pericardium may be suspected from certain signs; otherwise, diagnosis even within probable limits would in many cases be an impossibility. Infants and children who have withstood an attack of pericarditis, especially of the rheumatic form, are very prone to contract this form of pericarditis. In most cases it causes myocarditis of a progressive type; hence the importance of understanding the condition. Hypertrophy of the heart, atrophy of the heart, or dilatation of that organ may accompany adherence of the pericardium.

The **symptoms**, especially in the rheumatic cases, develop late in the disease when myocarditis supervenes. The condition may prove fatal by progressive affection of the cardiac muscle. One of my cases, of rheumatic origin, showed post mortem no valvular lesion. There were complete obliteration of the sac and extreme dilatation. The symptoms are at first negative. There may be a friction sound

or a roughening of the cardiac sounds at the base. There is in some cases a drawing inward of the apex area of the chest at the xiphoid cartilage. A wave-like undulation of the cardiac area with an increase of cardiac dullness is sometimes found. There may be persistent asystole not controllable by digitalis (Sée). In my cases there were angina, a persistently high pulse with an increase in the number of respirations, and in the last stages, all the symptoms of non-compensatory dilatation of the ventricle which are seen in valvular disease. There may be a mitral systolic murmur simulating that seen in valvular disease. In spite of all these symptoms, it is rarely possible to make a positive diagnosis during life.

### HYPERTROPHY AND DILATATION.

Cardiac hypertrophy and dilatation, combined or singly, and without any valvular lesion, occur in isolated cases in childhood. The condition is rare before the fifth year. A number of cases occurring between the fifth and the tenth year have been reported. If hypertrophy alone is present, it may affect the left ventricle only, or both ventricles. Dilatation usually affects first the right ventricle and then the left. The condition develops as a result of toxæmic influences, in the acute infectious diseases, such as scarlet fever, pneumonia, diphtheria, and typhoid fever.

Hypertrophy with or without dilatation is one of the sequelæ of acute or chronic nephritis. The nephritis complicating scarlet fever is frequently the cause of cardiac hypertrophy with or without dilatation. Atheromatous conditions of the arterial system with diminution of the calibre of the aorta may cause hypertrophy with or without dilatation. Acute dilatation as a result of heart strain is unknown in children.

The **symptoms** are not characteristic. In the absence of all other heart lesions, the diagnosis of cardiac hypertrophy or dilatation is made from the physical signs. These do not differ from those found in the adult subject. The rational symptoms also resemble those of the adult. In dilatation of the heart, there are the irregular heart action, the dyspnoea or orthopnoea, the pallor of the surface, cyanosis, and in the later stages swelling of the liver and spleen. Transudates in the pleural and abdominal cavities are apt to occur toward the close. Sudden death has occurred in some cases of dilatation of the acute variety. In hypertrophy, the symptoms closely resemble those just detailed. At the bedside, the diagnosis of hypertrophy, of dilatation, or of both, must of necessity rest on the physical signs.

The **treatment** varies with the nature of the primary disease (nephritis or toxæmia) present. The nephritis should be treated



and the heart will take care of itself. If there is an infectious disease, such as typhoid fever, diphtheria, or scarlet fever, both the heart and the primary affection should be treated.

#### LEADING AUTHORITIES REFERRED TO IN CHAPTER VII.

- Ausset, E.*: *Maladies des Enfants*, 1898.  
*Fleischman, L.*: *Klinik der Pädiatrik*, 1875.  
*Guttmann, P.*: *Untersuchungs Methoden*, 1886.  
*Henschen, S. E.*: *Ac. Herzdilatation*, Jena, 1899.  
*Heubner, O.*: "Ueber Chorea," *Verhandl. d. gesellschaft der Kinderärzte*, Hamburg, 1901.  
*Hochsinger, Carl*: *Auscultation des Kindlichen Herzens*, Wien, 1890.  
*Jürgensen, T.*: "Endocarditis," *Nothnagel's Spec. Path.*, 1900.  
*Jacobi, A.*: "Functional and Organic Heart Murmurs," *Med. News*, 1900.  
 ———: "Acute Rheumatism in Infancy and Childhood," *Amer. Clin. Lectures*, 1879.  
*Keating and Edwards*: "The Pulse in Childhood," *Archives of Pediatrics*, 1888.  
*Musser, J. H.*: "On the Disappearance of Endocardial Murmurs," *Brit. Med. Jour.*, 1897.  
*Mendelssohn, M.*: "Der Einfluss des Radfahrens," Berlin, 1896.  
*Packard, F. A.*: *N. Y. Med. Jour.*, June, 1899.  
*Sahli, H.*: *Topographische Percussion*, Bern, 1882.  
*Soltmann*: *Herzgeräusche im Kindesalter*, 14te *Verhandl. gesell. Kinderärzte*, 1897.  
*Sansom, A. E.*: *Diagnosis of Diseases of the Heart*, London, 1892.  
*Ullman, J.*: "The Tonsils as Portals of Infection, with Literature," *Medical News*, 1900.  
*Vierordt, H.*: "Die Angeborenen Herzkrankheiten," *Nothnagel's Spec. Path.*, etc., 1898.  
*Walshe*: *Diseases of the Heart*, London, 1873.  
*Weill, E.*: *Maladies du Cœur des Enfants*, Paris, 1895.

## CHAPTER VIII.

### DISEASES OF THE NERVOUS SYSTEM.

#### METHODS OF DIAGNOSIS.

##### Lumbar Puncture.

LUMBAR PUNCTURE was first practised by Quinke. It is to-day one of the most useful adjuncts to the methods of diagnosis in acute and chronic forms of cerebral and spinal disease. Its future usefulness as a therapeutic measure is not clearly established, but will probably lie in relieving symptoms due to pressure, and removing the excess of inflammatory exudate in the various forms of meningitis.

##### The Normal Cerebrospinal Fluid.

Normal cerebrospinal fluid is a clear colorless fluid having a slightly alkaline or neutral reaction. Its specific gravity varies from 1007 to 1009. It contains from 0.05 to 0.1 per cent. of albumin (Quinke, Rieken, Pfaundler), and because of the presence of sugar has a slightly reducing action on copper. It does not coagulate spontaneously. If centrifuged, a microscopic sediment of a few endothelial cells and leucocytes may be obtained. The cerebrospinal fluid is normally under a pressure of from 5 to 35 millimetres of mercury. The pressure in infants is lower than that in children. The causes of the variations of pressure and the nature of the conditions under which they occur have not as yet been determined. Respiration causes a deviation of fully 6 millimetres of mercury in the manometer column.

**Abnormal Conditions.**—The cerebrospinal fluid will in pathological states vary in respect to specific gravity, composition, appearance, and in the amount of sediment contained. The pressure in the subarachnoid and cerebrospinal spaces will also vary in different forms of disease.

The **specific gravity** in tuberculous meningitis varies from 1003 to 1011 (Lenhartz), in cerebrospinal meningitis from 1005 to 1012 (Pfaundler).

The **gross appearances** of the fluid obtained by lumbar puncture may be changed by the admixture of blood. Blood may come from the puncture wound or may have been in the canal previous to puncture as a result of a hemorrhagic pachymeningitis or of some form of cerebrospinal meningitis, traumatism, or apoplexy with



rupture into the ventricles. The wounding of veins either in the tissues or in the cauda equina may cause the admixture of blood. The quantity of blood may be just sufficient to tinge the fluid or the blood may be almost pure. It is not possible to determine whether the admixture of blood is or is not the result of accidental puncture of a vessel unless, as in pachymeningitis or traumatism, light is thrown on the matter by the history of the case and the presence of blood on repeated puncture. The accidental admixture of blood is unfortunate, since it obscures the microscopical diagnosis. The hemorrhage into the spinal canal is never alarming or of serious import.

**Tuberculous meningitis** changes the gross appearance of the fluid obtained by lumbar puncture. The fluid may be quite clear, exceptionally cloudy, opalescent, or in rare cases purulent. As a rule, however, it is clear in the early stages of the disease and cloudy in the later period. If the test-tube is held in a strong light, there may be seen, in a clear or cloudy fluid, myriads of highly refracting particles resembling the motes in a sunbeam (Moser, Bernheim, Pfaundler). The appearance is quite characteristic. It was first explained by Lichtheim, as the result of spontaneous coagulation. If a test-tube of the fluid obtained by lumbar puncture is placed in the upright position in an ice-box, there is found after twenty-four hours, a fully formed cobweb-like, funnel-shaped coagulum, beginning a little below the surface of the fluid and extending downward, the broader part of the funnel being above. According to Pfaundler, this coagulum is of diagnostic import. I have relied on its appearance in fluid which was not contaminated with blood, and found it of great value. The formation of the coagulum begins after the fluid has stood for two hours, and is fully completed by the following day. It is usually found from eight to twelve days before death.

**Suppurative Meningitis.**—In this form of meningitis, the fluid obtained by lumbar puncture is purulent, opalescent, grayish-white, grayish-yellow, or brownish (hemorrhagic). Exceptional cases give a clear fluid. There may be a spontaneous coagulum resembling that seen in tuberculous meningitis.

**Epidemic and Sporadic Cerebrospinal Meningitis.**—In the early stage of this disease, the fluid may be quite clear with suspended microscopic sediment. It may also be cloudy or thick, creamy or bloody. It may at first be clear, and later in the disease become purulent (Councilman).

**Chronic Hydrocephalus.**—This gives a clear fluid with no suspended particles visible to the eye, although microscopically there may be leucocytes. Pfaundler in one of his cases obtained a fluid which was cloudy because of the admixture of leucocytes.

Tumor of the brain gives a clear fluid. I have had a case of this kind.

**Sediment.**—This feature will be fully discussed under the sections devoted to Tuberculous Meningitis and Cerebrospinal Meningitis.

The **pressure** under which the cerebrospinal fluid is retained in the subarachnoid space and in the spinal canal is increased in the various forms of meningitis. This is especially true of tuberculous meningitis, in which the pressure may reach 110 m.m. of mercury. In this disease the pressure increases from the initial period to that of pressure symptoms, and diminishes toward the close of the disease—the stage of paralysis. Ventricular involvement gives the highest pressure figures. The following figures are taken from Pfaundler's tables :

First stage . . . . .	48 m.m. of mercury.
Stage of pressure . . . . .	52 m.m. " "
Stage of paralysis . . . . .	24 m.m. " "

In suppurative meningitis, the pressure varies from 10 to 37 m.m. of mercury ; in cerebrospinal meningitis, from 24 to 50 m.m. ; in hydrocephalus, from 6 to 60 m.m. ; in tumor of the brain, from 3 to 52 m.m. (Quinke, Slawyk, Pfaundler).

The presence of an increased amount of albumin in pathological states has been noted by Wentworth, Quinke, and Pfaundler. In tuberculous meningitis it may reach 0.3 per cent. ; in purulent meningitis, 0.6 per cent.

### The Operation of Lumbar Puncture.

The **instrument** consists of a trocar and canula such as is employed in tapping cavities. The best form of instrument has a handle sufficiently large to be grasped firmly (Fig. 136). The canula should be at least one millimetre in diameter. It is not necessary to use a manometer. In infants, the tenseness of the fontanelle is a rough guide in estimating the pressure in the subarachnoid space.

**Place of Puncture.**—The puncture is made in the space between the third and fourth or the fourth and fifth lumbar vertebrae. This point is obtained by palpating the crests of the ilium ; an imaginary tangent to these crests strikes the fourth space. The space above this imaginary line will, as a rule, be found to be the third space. Puncturing the canal in the space between the sacrum and coccyx or in the lower sacral space offers no advantages either anatomically or from a diagnostic standpoint.

**Method.**—Local anaesthesia only is necessary. The back of the patient is carefully scrubbed with green soap, then washed with alcohol and ether, and finally with sublimate. The patient is laid on either side according to the convenience of the operator. The spine is curved so that the spinous processes may be distinctly seen and palpated (Plate XVIII.). No considerable pressure should



PLATE XVIII.



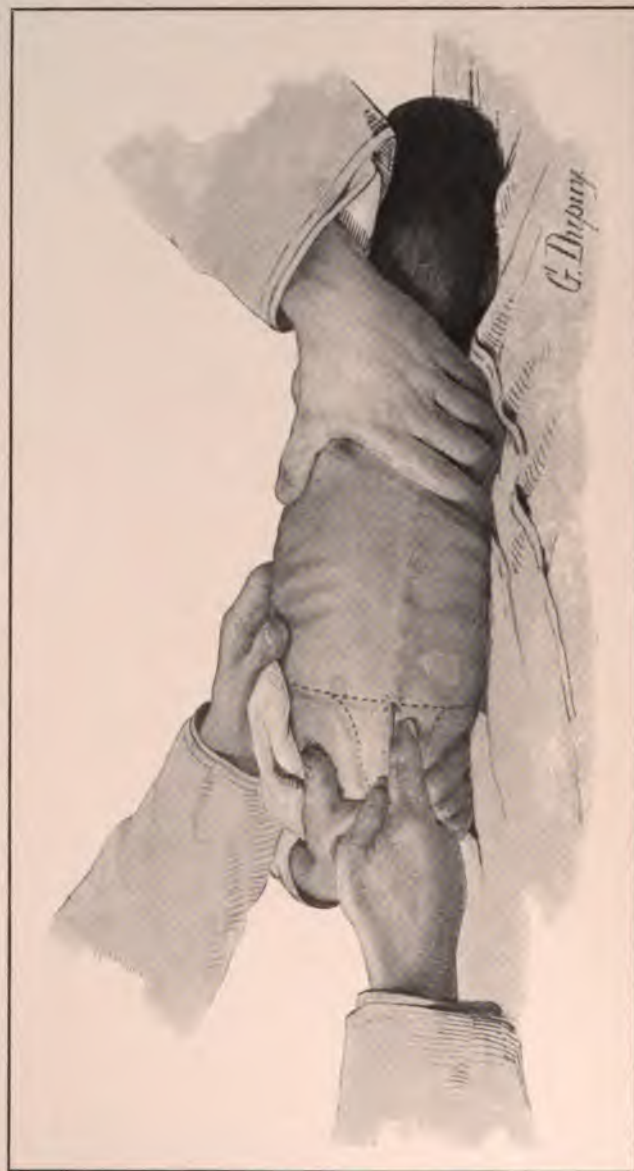
Operation of Lumbar Puncture. Method of holding the patient. Dotted lines show topography of the parts and the manner of finding the proper point for puncture.







PLATE XIX.



Operation of Lumbar Puncture. Method of introducing the trocar and cannula.



be brought to bear on the neck, since in cerebrospinal meningitis or in the basilar form of meningitis in which there is opisthotonos, serious injury to the neck may be caused. The spine is curved from the shoulders and pelvis. The needle, having been previously boiled, is introduced in the median line between the spinous processes and is directed upward (Plate XIX.). When it is in the canal, it is perceived that there is a lack of resistance, and that the point of the instrument is free. The canula is withdrawn and the first drops caught in a sterilized test-tube. A second test-tube is substituted for the first after a few drops of bloody fluid have been allowed to flow out, and from 10 to 50 c.c. of fluid are withdrawn, the amount varying with the pressure. If it flows drop by drop, 20 c.c. are sufficient for diagnostic purposes and also to relieve the pressure. If there is opisthotonos and the fluid does not flow well at first, cautious straightening of the neck will facilitate the outflow. In infants, the fontanelle is a good guide in gauging the pressure. As soon as a few cubic centimetres of fluid have been withdrawn, the fontanelle will be felt to be considerably flattened and relaxed or even depressed. Heubner has withdrawn 100 c.c., but the removal of such large quantities is unnecessary and may be followed by hyperpyrexia and collapse. I rarely withdraw more than 20 c.c. If there is a dry tap, the canula should be withdrawn and a second attempt made on the following day. A dry tap may be caused by a fibrin clot or by the falling of the cauda equina in front of the opening of the canula. The fluid may be viscid and refuse to flow. In that case the fluid should not be aspirated with a syringe, since in the experimental laboratory this method has been proved to be hazardous. If carried out as above directed, I have never seen any ill results from the operation of lumbar puncture. After puncture, the canula is rapidly withdrawn and the wound dressed with iodoformized gauze.

**Indications for Lumbar Puncture.**—Lumbar puncture is performed in the various forms of meningitis for diagnostic purposes, to determine the character of the fluid. It is not always an easy task to decide whether it should be resorted to. The decision is especially difficult in private practice, where the procedure is regarded with dread. Any marked disease of the lungs should first be excluded. In many cases of pneumonia the cerebral symptoms are marked. Only very marked symptoms of cerebral press-

FIG. 136.



Trocar and canula for performing lumbar puncture.

ure and the suspicion of pneumococcus meningitis should cause the physician to resort to puncture in order to fix the diagnosis. It is best not to perform it while the lesion in the lung is markedly active. Cases of tumor of the brain should not be subjected to puncture.

Indefinite cerebral symptoms, such as headache, restlessness, and convulsions of a general character, are not indications for puncture. I have seen cases of meningitis of the cerebrospinal type which gave few symptoms of disease, there being indefinite sopor, general muscular weakness with delayed reflex at the knee and marked emaciation, but no marked rigidity of the neck.

In doubtful cases I refrain from puncture. Cases with meningeal symptoms, in which there is a history of a blow, are proper subjects for puncture, since it is necessary to differentiate between meningitis, and abscess of the brain. In the various forms of purulent meningitis, symptoms of pressure such as convulsions, and signs of suppuration such as chills, are indications for puncture. After the first puncture is made and the diagnosis fixed, I do not in tuberculous meningitis repeat the operation.

The indications for puncture in other diseases such as hydrocephalus will be discussed under the headings of the various affections.

### INFANTILE CONVULSIONS.

(*Eclampsia.*)

Convulsions are a series of violent clonic contractions of a number of muscles or of the muscles supplying one limb. There is always more or less tonic spasm. The convulsions are paroxysmal, and are accompanied by loss of consciousness. In this section, only the acute convulsions of infancy and childhood will be considered. They should be sharply differentiated from certain affections, such as laryngismus, tetany, or epilepsy accompanied by spasms, although all these are classed as forms of convulsion. The acute convulsions of infancy and childhood are acute symptomatic phenomena. They occur chiefly during the first half-year of life. Fully four-fifths of the cases occur before the end of the second year (Kassowitz). They are uncommon after this period, but a child that has had convulsions in infancy is likely to have them on the slightest provocation up to the fifth year.

The pathogeny of convulsions in infancy and childhood is the same as in the adult. The explosions are due to irritation of the centres in the ponto-bulbar junction or in the area of Rolando (Hughlings Jackson). The starting-point of every convulsion is the ganglion cell (Peterson). The peculiar constitution and unstable state of the nervous system in childhood make children particularly sus-



ceptible to the action of the agents (toxins) which incite convulsions. It is not known whether inherited neurotic tendencies are powerful at this early period, or whether alcoholism or epilepsy in the family really influences the occurrence of purely acute convulsions. Rachitic children are particularly subject to convulsions, because the cranial bones are the seat of hyperæmia and softening (Kassowitz, Elsässer). The motor areas of the brain are supposed to be in a state of constant irritability.

The **etiology** of acute convulsions is very diverse. The majority of the convulsive seizures of infancy and childhood occur in connection with the infectious diseases, and at the onset of those affections. The explosion appears to be caused by the initial effect of the toxæmia and temperature reaction on the ganglion cell. Any infectious disease, such as acute amygdalitis, the exanthemata, influenza, pertussis, mumps, may be ushered in with a convulsion. Convulsions sometimes take the place of an initial chill in pneumonia and in malarial fever. Any infection from the gut, such as those due to indiscretions in diet, and gastro-enteric disease of any kind, may cause a convulsion. Children who eat an excessive quantity of meat are particularly subject to these seizures. Poisons circulating in the blood or a traumatism caused by a fall on the head are apt to cause convulsions in susceptible children; uræmia frequently causes convulsive seizures. In acute or chronic nephritis, the urine should always be tested. Dentition is frequently mentioned among the causes of convulsions. Since dentition in a normal infant is devoid of symptoms, it is stretching theory to ascribe infantile convulsions to irritation of the trigeminal branches. The acceptance of this theory might cause some serious condition, of which the first indication is an eclamptic seizure to be overlooked. A severe hemorrhage causing an acute cerebral anæmia may give rise to a convulsion, as may also morbid growths of the brain or cord. Convulsions of the latter form are hardly to be included in the practitioner's conception of infantile convulsions of the acute type.

**Morbid Anatomy.**—Kussmaul and Tenner have demonstrated that there is an acute anæmia of the brain during convulsions. On the other hand, it often happens that the convulsion is the cause of the bursting of a cerebral vessel. In such cases, the signs of cerebral surface hemorrhage are present at autopsy. In other cases, although death has occurred during a convulsion, nothing is found post mortem but an œdema of the brain substance, of doubtful origin.

**Symptoms.**—The majority of convulsive seizures in infants and children are single. In certain cases the convulsions are repeated and extend over a prolonged period. The latter are not cases of simple acute infantile convulsions. The symptoms of acute eclampsia are sometimes so very slight as to be scarcely noticeable. Only a very observant mother will see a slight twitching of the lips and eyelids, a

momentary turning of the eye and cessation of breathing, or a momentary spasm of the whole trunk. The expression "internal convulsion," so frequently heard, evidently denotes these slight eclamptic seizures. The genuine convulsion comes on without premonitory symptoms. There is a momentary tonic spasm of the body, the head turns to one side and upward, and there is a corresponding upward direction of the eye. Then follows a series of clonic spasms involving the upper and lower extremities, and lasting for some time. The patient froths at the mouth, the tongue is sometimes protruded and bitten, and there is involuntary passage of feces and urine. The whole paroxysm may last from half a minute to three minutes. The patient then passes into a stuporous condition or into sleep. During the spasm, the jaws are set and there is grinding of the teeth. There is cyanosis, on account of the spasm of the respiratory muscles and the interference with respiration. Not all convulsions end with the first paroxysm, although this is so in the great majority of cases. In cases in which the organism is peculiarly susceptible one paroxysm may succeed another rapidly. The child may be in a state of eclampsia for an hour, after which it may pass into the comatose state. The coma may be momentary or may merge into a sleep of variable duration. The end of the convulsive spasm is signalized by muscular clonic spasms decreasing in severity, until finally a long-drawn inspiration ends the attack.

**Diagnosis.**—It is very important to be able to distinguish between the various forms of convulsive seizures. Those occurring immediately after or within a few hours or days of birth have a different etiology from those just described. They may be caused by cerebral hemorrhage, and there will be symptoms after the convulsions, such as palsies, contracture, difficulty in deglutition, and prolonged coma. In these cases the convulsions are repeated. Atelectasis of the congenital variety may cause convulsions. The patients have slight or marked cyanosis, and, in the intervals, increase of respirations and signs of bronchitis and collapse of the lung.

Tumor and abscess of the brain, and meningitis, both cerebrospinal and tuberculous, may be ushered in by convulsions. In tumor, the convulsions are limited to the area in which the tumor or abscess is localized. In forms of meningitis, there will be the symptoms of that disease. Drugs and poisons may give rise to convulsions. The history of such cases will be of service. Cases of tetany and tetanus have convulsions in the course of the disease. In tetany there may be several convulsions in the course of twenty-four hours. Tonic spasm is the chief feature of the convulsion in tetany and tetanus. The clonic form distinguishes acute convulsions. In tetanus there is slowly increasing opisthotonos. In tetany the body may be lax in the interval, but there are rare cases of tetany which resemble tetanus in that there is rigidity in the intervals between the



spasms. In tetany the extremities have a characteristic position. In some cases of simple acute infantile convulsions, an increased irritability of the nerves and muscles to mechanical stimulus remains for days after the paroxysms. The Chvostek and Trousseau phenomena are found. Some authors have regarded these cases as cases of latent tetany. The diagnosis of the various epileptiform seizures will be considered in the section devoted to that subject.

The **prognosis** of acute infantile convulsions is generally good, but since death has occurred in these seizures, as well as cerebral hemorrhage, caution should always be exercised in predicting the immediate outcome. The patient having been once tided over the initial paroxysm, it may be confidently expected that it will not be repeated. In the presence of fever, it cannot be predicted what affection may follow the seizure. Primary seizures should not be regarded as forerunners of epilepsy. Many infants and children affected with convulsive seizures pass through later life without any sign of that disease.

**Treatment.**—The seizure is frequently over before the physician arrives. If such is the case and the infant is in the stage of stupor, it should not be disturbed unless there is high fever or a history of the patient's having eaten some irritating substance. It often happens that the paroxysm supervenes in the presence of the physician. The patient is placed on a bed, the clothes loosened, and a small object, such as the handle of a tooth-brush, placed between the teeth to save the tongue from injury. Nothing further is needed. The paroxysm is as a rule over in three minutes at most. If it persists or is immediately succeeded by another, the patient is placed in a warm bath, after which a few drops of chloroform are administered by inhalation to control the convulsions. A high rectal enema of the temperature of 110° F. (43.3° C.) is at once administered. I have in some cases continued the administration of chloroform for fully an hour. Caution should be exercised in its administration. If, after the seizure, the temperature is high, it is treated as indicated in the section on Infectious Diseases. Unless there is some contraindication, a full dose of calomel is administered as a routine procedure even if an enema has been resorted to. Should the child be restless, it is well after the convulsion to administer a dose of bromide of potassium in combination with chloral, either by mouth or rectum. In repeated convulsions the administration of these drugs during the seizures is of inestimable value.

For several years past I have used the postural treatment in primary convulsive seizures. The patient is placed with the head low, the buttocks raised, and the clothes loosened. I think that the paroxysms have been shortened by this treatment. It was suggested

by the theory that cerebral anæmia is the cause of the initial paroxysm. I have carried out this postural treatment without any ill after effects, such as hemorrhage. In a large number of cases of repeated convulsions, the postural treatment should be supplemented by chloroform inhalations.

### HYSTERIA.

Hysteria is a morbid state of the nervous system in which the primary derangement is in the higher cerebral centres. The lower centres of the brain, the spinal cord, and the sympathetic system may be secondarily disordered (Gowers). It is not a true disorder of childhood. Sixteen per cent. of all the cases of hysteria occur in youth (Steiner).

**Etiology.**—Hysteria is rarer in children than in adults, is more frequent in the female sex, and is more often seen in boys than in men. According to Briquet and Landouzy, 8 per cent. of all the cases occur in the first decade of life, and 50 per cent. in the second. The cases of the first decade, according to Barlow, generally develop at the age of six years. Cases are occasionally seen in patients of the age of three years. Heredity plays an important etiological rôle. Moral and mental influences predispose to development of the condition. Children of emotional antecedents are apt to be subject to the disease. Sexual disturbances or excesses (as masturbation in boys) are exciting causes. Abnormalities of the sexual organs, phimosis, and hypospadias, are apt to excite masturbation and resultant hysteria. In some subjects, any acute disease, such as pneumonia or typhoid fever, will develop latent tendencies to hysteria. Diphtheritic paralysis may eventuate in hysterical palsy (Gowers).

**Symptoms.**—The disease shows many variations and most diverse symptoms. The symptoms may be divided into psychic, motor, and sensory manifestations; or into the convulsive and non-convulsive forms of hysteria.

**Psychic or Mental Hysteria (Non-convulsive).**—In most cases of this class, the patients suffer from some mental strain. The attack begins with a paroxysm of crying or of laughing. The child then passes into a violent condition, striking at persons and tearing the clothes from its body. I saw a case of this kind in a boy eight years of age. He was very bright at school, but shunned the companionship of other boys. He masturbated. At times he was of a very loving disposition, at other times would refuse to do as he was told. The rebellion would terminate in a paroxysm of crying, followed by one of shrieking. The boy would tear his clothes and then calm down quite exhausted. Girls after undergoing some mental strain, such as is incident to a school examination,



become irritable, morose, and suffer from insomnia. They have laughing and crying spells and refuse nourishment. After a period of these symptoms they either recover or pass into a state resembling acute mania. Such children are nervous and are born of neurotic parents.

Hystero-epilepsy, catalepsy, or trance symptoms may manifest themselves. These cases are rare in children, but Sachs and Steiner have seen them in children of mentally degenerate families.

Insanity, alcoholism, and chorea in the family predispose to the development of hysteria. These cases must be differentiated from those of true epilepsy.

**Motor Manifestations (Convulsive Forms).**—These occur in the form of hystero-epileptic attacks. After some mental excitement, a paroxysm beginning with a shriek will supervene, the sounds simulating a bark or a snapping sound. Contortions then supervene and the back is arched, as shown in Richer's drawings. During the attack, which may last for several minutes, there may be no evidence of consciousness. There may be a number of such attacks in the course of twenty-four hours. The patient may suddenly fall down and have contortions, and the attack may terminate in a crying spell. The patients sometimes tear their clothing and become violent. These convulsions are differentiated from true epilepsy in that there is no aura; they are preceded by emotional excitement. The onset is gradual and the patients emit noises of various kinds during the attack. The pupils are normal. There are ecstasy, extravagant movements, and tonic rigidity. The vesical and rectal reflexes are normal. The patients do not bite the tongue, and rarely injure themselves; the loss of consciousness is temporary or imperfect. There are in hysteria irregular twitchings of the extremities and a repetition of one specific movement, such as retraction of the head. The spell or paroxysm ends in a crying or laughing fit, or the patients become melancholic.

Among the manifestations of hysteria in children is the so-called hysterical stricture of the œsophagus, or globus hystericus. There may be spasm of the bladder, hiccough, and loss of voice. The latter is common among young girls. I have seen the children recover their voice under hypnotic suggestion. Hysterical children may, even at the early age of five years, pass under hypnotic suggestion, into a trance-like state. Whether diarrhœa can be caused by hysteria is in my opinion doubtful. I have seen true toxic diarrhœa in neurotic children diagnosed as nervous or hysterical. One case occurred in a boy of six years. Some young girls have attacks in which all varieties of poses are assumed in the nude state. I have seen such a case in a highly intelligent girl of nine years. During the morning bath, the child had a desire to assume the most grotesque poses.

The so-called epidemics of chorea are now known to be simple hysteria. Among these are to be classed the school epidemics and the dancing mania of the Middle Ages.

There may not only be convulsive movements, but also absolute paralysis of single muscles or of a group of muscles. Hysterical paralyses as a rule follow no anatomical distribution. They are distinguished from true palsies by the lack of change in the electrical reactions and in the condition of the deep reflexes. The sphincters are normal. Paralyses, such as those due to neuritis or poliomyelitis, may supervene in a hysterical subject.

The **disturbances of sensation** include hyperæsthesias and anæsthesias. These do not differ essentially from similar conditions in the adult subject. There may be hyperæsthesia in the region of the ovary, or in the skin over the vertebral column. Areas of irritation may cause paroxysms. There are hysterogenic zones which are not hyperæsthetic (Sachs). Anæsthesia, partial or general, is more frequent. There may be absolute anæsthesia to all sensation. There may be blindness in one eye or hemianopsia, deafness, or loss of taste or of smell. Vision may be affected as above described, or there may be photophobia and diminution of visual perception; the retina may be insensible to light, and there may be limitation of the field of vision or temporary bilateral loss of sight.

There are in children cases of anorexia which supervene with vomiting after some nervous strain. I have seen this occur in children who were beginning some course of study. In one case it came on in the morning just before the child started for school. With suspension of school duties, the vomiting ceased. The so-called phantom abdominal tumor seen in rare instances among children may be traced to a hysterical cause. In very young girls I have frequently seen forms of palpitation with cardiac anguish which seemed to be hysterical. Steiner describes these forms of tachycardia. In these cases there is not only absence of cardiac lesion and signs of Basedow's disease, but spinal hyperæsthesia may be elicited.

**Diagnosis.**—Sensitiveness to pressure over the vertebral column is one of the most frequent stigmata of infantile hysteria (Steiner). Epigastric tenderness is less frequent than among adults. Hyperæsthesia is less marked in childhood than later in life, but is more common than anæsthesia. Jolly says that deep analgesia is rare. Of especial interest in its relation to diagnosis is the fact, that ocular symptoms, such as diplopia, may be present morning and evening. Paralysis may appear and disappear. There are forms in which there may be tachycardia or bradycardia, but during excitement the rhythm of the heart may be normal. Cases have been described in which the headaches, ptosis, and facial palsies simulate the symptoms of tuberculous meningitis. Study alone will clear up such obscure cases.



**Duration and Course.**—The symptoms of hysteria are not necessarily permanent, but are likely to recur after excitement or nervous strain of any kind.

The **treatment** of hysteria in children is based on the same general principles as in the adult. The child is, if possible, removed from exciting surroundings. Studies are regulated and bad habits, such as masturbation, are, if possible, corrected. The effect of good food and outdoor life is marked. Hydrotherapy and massage achieve their greatest triumph in this affection.

## TETANY.

(*Tetanilla*; *Arthrogryposis*.)

Tetany is an intermittent or persistent, more or less painful, tonic spasm of groups of muscles of the upper and lower extremities.

**Forms and Frequency.**—John Clark in 1815 described this disease in children. Trousseau, Baginsky, Chvostek, Erb, Escherich, and Ganghofner have completed its symptomatology. In children it is most common from the fourth to the twentieth month. There are two distinct forms. In the first, the contractures are intermittent, and come on at intervals, the patients being free from muscular spasm in the intervals. The second form, now accepted by the majority of writers as the same affection as the former, is that in which the contractures are persistent.

The **etiology** of this affection is still very obscure. It occurs most frequently in the winter and early spring. In my experience in an ambulatory clinic, it was customary to see these cases appear in groups in the early spring months. The affection is seen under the most diverse conditions. Fully 63 per cent. of the cases are rachitic (Fischl). The percentage of rachitis must, of course, vary in different countries, but the cases coming under my notice have been chiefly of that character. The condition is not, as is frequently supposed, a rare one. I have regularly seen a number of these cases yearly. Many cases of tetany are not recognized as such by the physician. Cold, entozoa, infections of the gut, chronic intestinal disturbances of all kinds, rachitis, an enlarged thymus (Escherich), have all in turn been regarded as etiological factors. On the other hand, the general trend of opinion is to attribute the affection to a toxæmia probably originating in the gut and expending itself on the peripheral motor nerves. Fully 73 per cent. of Fischl's cases had shown intestinal disturbances. The fact that the condition occurs in early infancy and in some respects resembles a normal state, to be described later, will account for its being frequently overlooked by the physician.

**Morbid Anatomy.**—No definite account of the changes in the

nervous system or elsewhere has as yet been given. Langhans has described a peri-arteritis and phlebitis in the white commissure and cervical portion of the cord. Gowers, without any positive data, assumes that there are some changes in the motor cells of the cord which cause the increased irritability of the peripheral motor nerves. Fischl in a recent article has published the post-mortem changes in his fatal cases. He makes, however, no comment on them. He found hydrocephalus interna and externa, œdema of the brain and meninges, tuberculosis of the brain, hemorrhagic infiltration of the cerebellum and meninges, chronic intestinal catarrh, and bronchopneumonia. The affection occurs under the most diverse conditions.

The **symptoms** consist of muscular contractures and phenomena connected with the peripheral motor nerves, which are known as Trousseau's phenomenon, Chvostek's facial symptom, and Erb's signs of increased electrical excitability of nerve and muscle.

**Muscular Contractures.**—These come on without any premonitory symptoms. The infant or child may have been in good health, or may have been suffering from intestinal disturbance. There are two distinct forms of contracture in infants, in one of which the hands and arms take the position assumed in driving horses (Plate XX.). The arms are pressed against the thorax, the forearms flexed on the arms, and the fingers tightly flexed over the thumb into the palm of the hands. The hand itself is strongly flexed on the forearm. The lower extremities may be adducted toward the median line, the thighs flexed on the abdomen, and the legs on the thighs. The feet are as a rule extended in the equinus position and the toes overflexed on the plantar aspect of the foot, the whole foot being slightly curved inward. After the contractures have lasted some time, there is œdema of the tissues over the dorsum of the foot. In the second set of cases the fingers are overextended, as shown in Fig. 137. The arms and lower extremities also take the position of flexion. These contractures are painful; the patient cries as if in great pain when an attempt is made to straighten the fingers or extremities. There may be a temperature of two or three degrees. The contractures may diminish, and there may be an interval in which the only symptoms are such as may be attributed to the increased mechanical and electrical irritability of the peripheral nerves. There may also be eclampsia. The eclamptic attacks are very dangerous. I have lost 2 cases in such seizures. Other muscles, such as the abdominal or thoracic, may be the seat of contracture. In the latter case there may be cyanosis.

I have seen cases in which all the muscles of the body were involved very much as in tetanic conditions. In one case there were stiffness of the muscles of the neck and loss of consciousness. Trismus is rare, and certainly does not occur at the outset, as in tetanus. The muscles of the face may be subject to contracture.



PLATE XX.



Tetany. Infant nine months of age. Shows the driving position of the fingers, hands and arms, overextension of the feet and flexion of the toes.





The brow is wrinkled, and the face has an anxious expression. If the muscles over the zygoma are tapped, there is an instantaneous contracture or spasm of the orbicularis palpebrarum. In some cases, if the muscles of the face or the forehead are tapped, there is an instantaneous contracture of the muscles of the face, and sometimes of other muscles of the body. This is called the facial phenomenon of Chvostek. If the nerves and arteries at the bend of the elbow are compressed, the characteristic tetany position is

FIG. 137.



Tetany. Extension of the fingers, flexion of the arms, flexion of the toes. Child, eighteen months of age.

produced in the muscles of the hand and fingers. This phenomenon was first noticed by Trousseau, and bears his name. Erb established the fact that there is increased irritability of nerve and muscle to the faradic and galvanic current. If the muscles or nerves elsewhere in the body are tapped, or if pressure is brought to bear at the point of exit of the nerve-trunks, there is an excessive irritability to this mechanical stimulus. The knee reflex is increased.

**Duration.**—The disease may last a few hours, days, or weeks. In many cases the contractures disappear for a time, leaving the patient perfectly free from symptoms. They may return in all their original severity. The attacks leave the peripheral nerves in a condition of increased excitability. In such cases both the Chvostek and Trousseau phenomena may be present.

The **diagnosis** of fully developed tetany is based on the presence of muscular contractures, of increased mechanical irritability of the peripheral nerves (as evinced in Chvostek's symptom), and the presence of Trousseau's phenomenon. There are cases of tetany in which the facial symptoms are lacking. On the other hand, I have, in cases in which there was laryngospasm without contractures, obtained both the facial and Trousseau phenomena.

**The Relationship of Laryngospasm to Tetany.**—Escherich, his pupil Loos, and also Ganghofner, have recently called attention to the fact that laryngospasm is present in a certain number of cases of tetany. They also found that cases of laryngospasm which did not present contractures, did show the facial phenomenon of Chvostek and the Trousseau symptom. They concluded that laryngospasm was a manifestation of tetany, whether the muscle contractures were present in the extremities or not. Their observations have been amply confirmed, but not all observers are as yet willing to accept laryngospasm without contractures of the muscles of the extremities, as true tetany. The views of Kassowitz and Hochsinger are at variance with those of Escherich. They consider rachitis the fundamental cause of laryngospasm, if not of tetany.

The term *latent tetany* has been applied to those cases which show no muscular contractures or laryngospasm, but in which the facial Trousseau or Erb phenomenon may be elicited.

**Prognosis and Mortality.**—The prognosis in the sporadic cases is very good. Parents should be cautioned in regard to the excitability of the patient and the possibility of eclampsia, with its fatal consequences. I have lost 2 cases in convulsions. The persistent cases may be complicated with other affections, such as tuberculous meningitis. If such is the case, the outcome is, as in the primary disease, fatal. Epidemics in hospitals for children present unfavorable features; Escherich lost 37 per cent. of his cases.

**Treatment.**—The bowels should first be evacuated. Calomel is given in grain  $\frac{1}{2}$  (0.03) doses two or three times daily. If there is any disturbance of the gut, the patient is given a high enema once a day. Milk is suspended until the movements take on a more favorable appearance. The infant is kept under the influence of the mixed bromides of potassium, sodium, and ammonia. If there is eclampsia or increased irritability, a warm bath is given at least once a day. The patient is kept quiet and not disturbed much. No attempt to straighten the limbs should be made, since it causes pain.



**CATALEPSY.**

Epstein has recently described a condition in children closely resembling a similar affection in the adult. He has described it as catalepsy occurring in infants poorly nourished and rachitic. The ages of his cases ranged from eighteen months to three and one-half years. Epstein believes there is a disturbance of the psychomotor functions. The phenomenon was observed by him chiefly in the lower extremities. Either extremity on being lifted into the air would stay there for a length of time in any position of flexion or extension in which it was placed. This phenomenon was not present during sleep, nor was it accompanied by any muscular rigidity or increase of mechanical or electrical irritability of the peripheral nerves. I have met a marked case of catalepsy following an attack of typhoid fever in a boy of four years. The hands, arms, and lower extremities would remain for long periods of time in the position in which they were placed. The boy would sit for long periods staring ahead, without winking the eyes.

**MYOTONIA.**

Myotonia physiologica neonatorum is a term applied by Hochsinger to the normal tendency of the newly born infant to flex the fingers, arms, and lower extremities. There is a slight rigidity which is a hypertonicity of the muscle, and which lasts until the third month. The position closely resembles that of the extremities of the fœtus in utero. The myotonia is exaggerated if the infant becomes ill with any intercurrent affection, such as syphilis. The condition cannot be mistaken for tetany if the differences between the normal and the abnormal states of the peripheral nerves are borne in mind.

**CONGENITAL STRIDOR OF INFANTS.**

(THOMSON.)

This rare condition has for a long time been classified by writers as a mild form of laryngismus stridulus. I have seen one case in which there was also laryngismus. The affection is a distinct one, is generally congenital, and appears soon after birth. Some years ago, I presented a case of the kind before the Pediatric Section of the Academy of Medicine of New York. Since then I have seen a number of cases, and have records of four which I studied. Thomson has fully described and studied the affection. The infant is usually in other respects normal, but I have seen the condition in infants with signs of rachitis. The ages of the patients varied from nine weeks to twelve months. In one case there was a history of

attacks of laryngismus stridulus, occurring shortly after birth. In most of the cases, the symptoms were noticed soon after birth. The respiration is more or less noisy, being sometimes scarcely audible and at other times so loud as to be heard at some distance. Inspiration is accompanied by a peculiar croaking, grunting noise. As a rule, expiration is noiseless, but it may be accompanied by a grunting sound, there being short intervals in which no sound is heard. The infants are not at all disturbed by the condition. They sit and play, emitting this peculiar croak while breathing. In mild cases, nothing is seen in the thorax. I have, however, seen the drawing inward of the suprasternal region which Thomson describes. In one case the noise was louder at night. If the stethoscope is held over the situation of the vocal cords, it will be ascertained that the sound is produced in the larynx and not in the pharynx.

The causation is obscure, but the theory advanced by Thomson is probably correct. He surmises that there is an ill-coördinated spasmodic action of the muscles of respiration, choreiform in character and similar to that present in stammering. Others have attributed this condition to the presence of an enlarged thymus (Variot). Toward the second year of life, the condition gradually disappears spontaneously.

### LARYNGISMUS STRIDULUS.

(Spasm of the Glottis.)

Laryngismus stridulus is a spasmodic functional nervous disorder of the glottis, also involving the muscles of inspiration and expiration.

**Occurrence.**—The affection is more frequent in boys than in girls. It is most common in the first year of life. The majority of the cases occur before the end of the second year. Kassowitz found 348 of 370 cases to occur before that time. It may occur in the newly born infant (Henoch, Kassowitz). Most of the infants and children affected by this disorder are subjects of rachitis and also show signs of craniotabes. Henoch estimates the frequency of rachitis at 75 per cent. Only one of the cases of Kassowitz did not show its signs. All but 48 showed craniotabes. On the other hand, Boral shows that 4 per cent. of all children with rachitis have laryngismus stridulus.

The **etiology** of this affection is obscure. Although rachitis is so frequent an accompaniment of the disorder, it may not yet be assumed that it is the exciting cause. Craniotabes, which is a part of the symptom-complex, has been regarded as the cause (Elsässer).

Escherich, Loos, Gee, and Ganghofner have placed laryngismus stridulus in the same category as tetany, and trace it to the same exciting cause. Reflex irritation from the stomach acting through



the vagus, is the theory of Baginsky. In many cases which have terminated fatally an enlarged thymus has been found. On the other hand, there have been *post mortems* which showed a rather small thymus and slightly enlarged bronchial nodes (Baginsky).

**Morbid Anatomy.**—No definite study has been made of the changes found in the fatal cases. Most cases show oedema of the brain and some fluid in the ventricles, rachitis slight or pronounced, the thymus small or enlarged, and the lymph-nodes slightly enlarged. The cases with enlarged thymus thus far published have not been convincing. Children with enlarged thymus die of other disorders, and without having had during life any symptoms of spasm of the glottis.

**Symptomatology.**—The spasm or paroxysm comes on suddenly. Without the least warning, the child throws the head back and stops breathing; the face becomes livid, the arms are flexed and the hands clenched. No respiratory movement takes place for a few seconds. There is then a long-drawn whistling or crowing inspiratory sound. This is the classical form of spasm of the larynx. The paroxysm may begin with a piping, inspiratory sound. Apnoea lasting for a varying length of time succeeds, and is followed by a loud or silent expiration. Apnoea may appear first, and be followed by several noisy explosive expiratory movements, which may be succeeded by several noisy crowing inspiratory sounds. The picture is usually that of spasm of the glottis as first described, in which the breathing stops entirely. The attack may come on during absolute quiet or during sleep, the onset of the attack causing the child to wake. The paroxysms may be brought on by excitement, a draught of air, or by pressure on the larynx. They are of all degrees of severity. Some infants show a form which is very disquieting. In a fit of crying the child takes a number of noisy inspirations and expirations, and then stops breathing, becomes cyanosed, clenches the hands, and threatens to pass into an eclamptic paroxysm (expiratory apnoea), when suddenly a deep inspiration occurs and the danger is passed. Some cases of the classical form have eclamptic seizures. There may be convulsions, especially in the form described as expiratory apnoea. In all of these cases there is the ever-present danger that the glottis and the muscles of respiration will fail to relax, thus causing death with convulsions. The number of attacks of spasm of the glottis may reach twenty or thirty a day, or they may be very infrequent, occurring only once every few days, weeks, or months. In all the forms, including the classical one just detailed, the spasm involves not only the glottis, but also the diaphragm and other muscles of respiration. The infants may show no symptoms after the paroxysms. On the other hand, some infants seem to be overcome and pass into a stupid state lasting for fully ten minutes (Henoch). It is difficult to estimate the degree of consciousness during an attack, but even in

the mildest forms there may be a momentary loss of consciousness (Henoch). Most cases show the facial and Trousseau symptoms of tetany and increased irritability of the peripheral nerves.

The **prognosis** of spasm of the glottis is good. The danger lies in the eclampsia, during which death may supervene.

The **diagnosis** is not difficult. There are all degrees of severity of the spasm, ranging from partial to complete closure of the glottis. In the latter form a rachitic infant in a paroxysm of crying is frequently heard to give several inspiratory crowing sounds without having any further symptoms. There is a species of inco-ordination. These cases may at intervals develop typical paroxysms. The parents should be warned of this possibility. The forms of spasm of the glottis which have just been described should not be confused with spasm or difficult breathing due to pressure of a retropharyngeal abscess or suppurating gland upon the larynx.

**Complications.**—Pertussis may complicate a case of spasm of the glottis. Cases thus complicated give a grave prognosis (Henoch). Tetany has been elsewhere mentioned as an accompanying condition.

**Treatment.**—**During the Attack.**—The infant is carried to an open window. A draught of air is allowed to blow in its face or a few drops of water are thrown in the face. This is done to excite a reflex relaxation of the glottis. The head should be held low, as in ordinary eclampsia. If relaxation of the glottis does not occur and convulsions set in, a few drops of chloroform may cause the muscles of respiration and those of the glottis to relax. Intubation and tracheotomy have been performed at this crisis, when the breathing threatened to cease permanently. If, however, as sometimes happens, the muscles of respiration are also involved, the paroxysm will occur with the tracheotomy tube in the trachea. Stück has published a case in which the insertion of a tracheotomy tube had not the least influence on the paroxysms. This is a very important observation, and raises the question of the propriety of intubating or performing tracheotomy. On the other hand, cases have been intubated and resuscitated with artificial respiration (Pott). In the intervals, the treatment should be chiefly directed toward the rachitis. The feeding should be carefully attended to; the infants should, if possible, be breast-fed. They should be shielded from the least excitement. Bathing in cold water has not in my experience been productive of good results.

## EPILEPSY.

Epilepsy is not a disease peculiar to infancy and childhood. It is discussed here simply to emphasize the peculiarities of the affection as seen in children. It is a true disease of the nervous system,



and has nothing in common with and no demonstrable relationship to infantile convulsions. Fifteen per cent. of the cases of epilepsy occur before the fifth year of life. Henoch has seen a case in an infant one year of age who had convulsions beginning with a cry and during which the infant bit the tongue. He describes another case in a child three years of age, in which the attack began with vertigo. In another case, in a child three years of age, the patient fixed a point and ran blindly toward it. The latter appears to have been a case of "procurive epilepsy."

**Etiology.**—According to Gowers, in two-thirds of the cases of epilepsy in children the parents are neurotic and hysterical. Chorea in the mother will often manifest itself in epilepsy in the child. Infantile palsy or traumatism is more frequently than heredity the cause of epilepsy. Epilepsy following slight palsy is likely to be mistaken for hereditary epilepsy.

**Symptoms.**—In children, as in the adult, there are no symptoms in the intervals between the attacks. Only such results of attacks as a bitten tongue or local traumatism are seen. There are, as in the adult, two distinct forms of epilepsy—grand and petit mal—between which there may be all variations participating in the peculiarities of both forms. In grand mal there is the aura, sensory or psychic; it is present in a large percentage of the cases in children.

Baginsky calls attention to a case in which epigastric pain was the aura preceding the attack. The other forms of aura are numbness and tingling of the extremities, general restlessness and irritability and auditory phenomena in which a peculiar cry of an animal is perceived. There may be a hissing sound. An aura referred to the sense of taste is very rare, and most neurologists do not make note of having found it in any case. In children the perception of peculiar odors just prior to the attack occurs as a form of aura.

After the aura, the attack begins with a cry followed by sudden loss of consciousness and tonic or clonic spasm of the muscles, which may be unilateral, general, or partial. The pupils dilate; there is spasm of the respiratory muscles and those of the jaw, as well as foaming at the mouth and biting of the tongue. The spasm then relaxes, the movements become first clonic and then intermittent, there is involuntary passage of urine and feces, and consciousness gradually returns, the patient passing into prolonged stupor and profound sleep. Some of these symptoms may be absent, but the loss of consciousness, dilated pupils, spasm, and the succeeding profound sleep are constant. In the majority of cases, the presence of any two of these will be sufficient for making a diagnosis.

**Convulsions.**—General convulsions indicate hereditary epilepsy. Convulsions may at first be partial, but in the majority of cases eventually become general. Partial convulsions indicate disease in the motor areas. The attacks taking the form of petit mal may

be so slight as to be mistaken for fainting spells. Such attacks may occur in young children. One of my cases was in a child five years of age. An epileptic spell is momentary; a fainting spell is gradual, there are no vasomotor disturbances, and the pupils do not dilate. Henoch and others record cases in which the children momentarily stop the occupation in hand, stare into vacancy, and then recover themselves without having any recollection of the interruption. In other cases there is an irritable attack or mild maniacal outbreak. In some cases the child passes into a state of mental confusion in which it performs acts unconsciously. Attacks of double consciousness or narcolepsia are rare in children (Sachs).

Attacks of grand mal are sometimes associated with a rise of temperature. A case recently came under my observation in which a girl of eight had as many as forty convulsive seizures in twenty-four hours. There was a slight rise of temperature which could not be traced to any cause other than the convulsions. Thomson and Oppenheim have shown that there are a concentric limitation of vision and a diminution of general sensibility for some time after the epileptic attacks.

**Diagnosis.**—Epilepsy must be differentiated from syncope, hysteria, post-hemiplegic convulsions, and tumor of the cerebrum. The peculiarities of an attack of syncope and hysteria have been dilated upon. The post-hemiplegic convulsions will, in the intervals, reveal the paralyzes and contractures with increase of deep reflexes. Attacks of convulsions caused by tumor are confined to groups of muscles if the tumor is in the motor area, and are combined with optic neuritis if the chiasm is directly or indirectly the seat of pressure.

With tumor, there are in the intervals peculiarities of the gait which aid in diagnosis.

The **treatment** of epilepsy is essentially the same in children as in the adult subject.

### PAVOR NOCTURNUS.

(*Night-terrors.*)

There are two forms of this affection—the primary or idiopathic and the symptomatic form. In both, the children retire to sleep and after an hour or two suddenly awaken from deep slumber with a shriek or cry. They are pale, greatly terrified, and grasp at the empty air. In incoherent, broken phrases they try to collect their thoughts. Some children see terrifying visions and either cling to the bystander for protection or try to get out of bed to escape an imaginary danger. After being quieted the children fall asleep, and when questioned the next morning have no distinct recollection of what has occurred. These attacks may



occur every night for days, weeks, or months. They rarely occur twice in the course of the same night.

The idiopathic form of this affection may occur in children who are naturally of a nervous temperament without any apparent exciting cause. I have seen it in children who were distinctly the opposite of nervous, and who were well nourished and good natured. The night-terrors may follow epilepsy or they may be so severe as to be the exciting element in precipitating an attack of chorea. Children sometimes have real hallucinations, which may be present even during the day (Henoch). It may, however, be said that hallucinations during the day are really not included in the idiopathic form. This affection occurs chiefly up to the time of second dentition. Forms of terror in older children are hysterical. Adenoids are supposed to be an etiological factor, but this is doubtful. It is only in the symptomatic form that children, after having committed some error in diet, awake with the symptoms above described.

The **prognosis** is good. The affection never precedes insanity. It subsides under treatment or disappears spontaneously.

**Treatment.**—In the symptomatic form, the meals should be so arranged that the lightest repast is that taken in the evening. In the idiopathic form, bromide of potassium is most useful. It is administered in one dose, an hour before retiring. The children should not be too active mentally during the daytime. Visitors should be restricted to certain hours. Play and sport in the open air are indicated. The school tasks of older children should be completed in the afternoon.

## CHOREA.

(*St. Vitus' Dance*; *Sydenham's Chorea*.)

Chorea is a nervous disease characterized by irregular involuntary movements or twitchings of some or all of the muscles of the body. It is accompanied by muscular weakness and mental disturbances. In some cases there is endocarditis.

**Classification.**—Chorea minor is an acute disease described by Sydenham. Chorea major is a hysterical disorder; under this heading are included the chorea electrica, and the dancing mania with rhythmical motions, of the Middle Ages.

Chorea insaniens is the fatal form of acute chorea minor.

Laryngeal chorea is a hysterical affection (Gowers).

Choreiform affections or pseudochoreas comprise the cases of tic convulsif of French writers and other forms of habit-spasm, local or general.

In addition there are forms of chorea which are symptomatic or secondary to infantile palsies. Huntington's chorea is a chronic progressive affection of a hereditary nature.

All these forms of chorea except chorea minor and insaniens should be excluded from the category of Sydenham's chorea.

The epidemics of so-called chorea, occurring in schools, are probably hysterical disorders which are the result of imitation and not true Sydenham's chorea.

**Frequency and Etiology.**—Chorea is more common among female than male children. Of 554 cases collected by Osler, 70 per cent. were of the female sex. It rarely occurs before the fourth year. Starr's statistics of 1400 cases show 8 at the third year. Cases are recorded as occurring in newly born infants, but are not accepted by all authors as authentic. The disease is most common from the fifth to the fifteenth year. Fifty per cent. of Starr's 1400 cases occurred before the tenth year, and 75 per cent. from the fifth to the fifteenth year. Of 83 cases of chorea occurring in my ambulatory and hospital service, 23 were of the male and 60 of the female sex. Ten children were under the age of five years, and 67 cases occurred from the fifth to the tenth year. Thus, the greatest frequency is at the latter period. Only one case occurred in a very young child (two and one-half years). The disease is found in children in all walks of life. Children of a nervous, ambitious temperament with a hereditary neurotic history are more prone to contract this disorder than those of a more equable disposition. It is therefore more common in towns and large cities than in country districts. In some cases there is a history of fright or traumatism, either immediately preceding an attack or coincident with its onset. It is as yet impossible to say, however, whether there is any relation between chorea and these occurrences. They may have some influence in developing latent tendencies to the disease. An attack will often be initiated by a scolding or chastisement on the part of parents. The spring months show the greatest number of cases, the least number occurring in the late autumn. There also appears to be a correspondence in the prevalence of cases of chorea and rheumatism at certain periods of the year (Osler, Lewis). The relation of a condition of lymphatism (adenoids or nasal catarrh (Jacobi)) to true Sydenham's chorea is not generally accepted. Errors of refraction in the eyes also seem to be a predisposing cause of the outbreak of choreic attacks. (de Schweinitz). These can scarcely be regarded as a direct cause of Sydenham's chorea, but acute articular rheumatism may be so considered. Rheumatism seems to run in families in which the children have chorea. Osler finds that 15 per cent. of his cases are of such families. Of the subjects of chorea, fully 21 per cent. show a history of rheumatism (Osler). These figures correspond more or less to the statistics of Townsend, 21 per cent.; Starr, 21 per cent. in 1400 cases; and my own cases, 18 per cent. Crandall gives the highest frequency of rheumatism in cases of



chorea (54 per cent.). In the majority of cases the rheumatism precedes the chorea (Sée). I have seen one case of chorea preceding an attack of rheumatism in a child four years old. I believe that, with cases of rheumatism of the acute articular type, there should also be included those of articular pains without swelling of the joint. The forms of rheumatism with chorea giving the so-called subcutaneous fibrous rheumatic nodules are rare in this country (Osler).

Chorea may complicate any acute infectious disease, such as scarlet fever, whooping-cough, measles, diphtheria, typhoid fever, and forms of sepsis. There are, however, no definite data of the exact relation, if there be such, between chorea and the infectious diseases. The theory that an attack of any of these diseases will cut short an attack of chorea is not borne out by clinical experience (Henoeh).

**Morbid Anatomy.**—The pathology of chorea is still incomplete and can therefore be merely indicated. Hyperemia of the brain and cord were found by Pye-Smith and Ogle. Anæmia and proliferation of connective tissue were recorded by Steiner. In the cases of Meynert there was hyaline degeneration of the nerve cells of the central ganglia. Flechsig mentions hyaline degeneration of the lenticular nucleus. Dana studied some cases in which he found hyperæmia of the brain, and degenerative changes in the walls of the bloodvessels of the white substance, with perivascular exudation and accumulation of leucocytes. Jackson has advocated the embolic theory (endocardial). At present there is a great preponderance of evidence in favor of the infectious theory. Berkeley found staphylococci in the blood in a fatal case of chorea. In another case, Naunyn found cladothrix in the meninges and endocardial vegetations. It is certain that just as rheumatism and endocarditis are infectious diseases, so chorea in many cases can only be understood on that theory. Cesaris-Demel has experimentally shown that the central nervous system is peculiarly susceptible to certain pathogenic micro-organisms and their toxins. The staphylococcus and its toxins when injected experimentally under the dura mater cause the formation of small foci of inflammation, and symptoms very similar to those of chorea.

**Symptoms.**—Children will at the outset of this disorder exhibit mild symptoms of nervous irritability, will be cross, have outbreaks of peevishness and temper, will drop things, and be generally careless in their habits. There is sometimes a history of night-terrors or morose crying spells. There is likely to be loss of appetite; headache is not uncommon, and there may be pains in the limbs or joints and general restlessness. The disease may begin in a certain set of muscles, or in the muscles of one-half the body and thence spread to the whole trunk. Of 301 cases of the statistics of Sachs, there was hemichorea or involvement of one set of muscles in

67. Of Starr's 1400 cases, 951 were general and 449 unilateral, the right side being affected more frequently than the left. When fully developed, the picture presented by these patients is so characteristic as to be easily recognized. On the other hand, the popular notion, so prevalent even among physicians, that every twitching is choreic, has led to grave errors. The following are the main symptoms:

**Motor.**—The twitchings usually begin in the right hand, only rarely in the legs. After a time there are incessant, irregular, awkward twitchings of all the muscles of the body, which are intensified by volition. If the child is directed to stand still, with the feet together and the arms and hands held out at right angles to the body, the motions are intensified. If it is told to close the eyes, there is a distinct swaying of the body. The movements are not only irregular, but awkward. The patients trip in walking, upset their food and drink, and cannot button their clothing with ease. As a rule, the muscular twitching ceases in sleep, but it may persist. The muscular power is weakened, although distinct paralysis does not occur. The muscle is more paretic than paralytic. Some children let the arm hang at the side. There is wrist-drop when the children are asked to hold out the arms. The tongue is affected in all cases. Sachs places much diagnostic value on the choreic movements of that organ. When children are asked to show the tongue, they will protrude the organ with a jerk, then withdraw it and twist it here and there in the cavity of the mouth. When the tongue is held out quietly, fibrillary twitchings in the organ may be detected. Electrical reaction or irritability of the muscles in chorea can be tested only when the disease is unilateral. In some cases there is no change. In others, according to Gowers, there is a distinct increase in the galvanic and faradic irritability of nerve and muscle. The muscles of the hands, face, and extremities are all involved in the twitchings of the voluntary muscles. The involuntary muscles, such as the cardiac muscle, are not affected. Their involvement has long been a matter of discussion.

**Disturbances of sensation** are not common. Children have the arthritic pains. Numbness, tingling, pricking, and anaesthesia of the pharynx are recorded. Attacks of multiple neuritis and epileptic seizures should be regarded as complications. The reflexes are not markedly affected. They may in rare cases be slightly diminished or increased (Hench). Any marked change in the reflexes may be traced to changes of an organic nature, in the cord. The occurrence of headaches or eye-strain as concomitant conditions has been referred to.

**Urine.**—The urine may contain albumin. Cases with nephritis as a complication have been reported (Thomas).

The **speech** is affected in 25 per cent. of the cases. The patients hesitate and mumble their words or there is difficulty of phonation

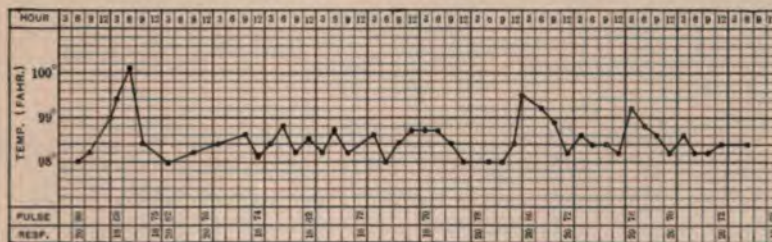


due to inco-ordinate action of the larynx. Laryngeal chorea, in which there is a distinct sound resembling a bark, is seen in rare cases. It is classified by Gower as a hysterical disorder, truly choreic. I have never met a case of the kind in a child. Deglutition may be affected because of the muscular inco-ordination.

The **cardiac symptoms** are the most important clinical feature of chorea. There is very little doubt, that in a fixed proportion of cases, rheumatism plays an important rôle and that the rheumatic poison, whatever it may be, expends its force upon the endocardium and pericardium. In 20 per cent. of the cases of Osler and in 12 per cent. of Starr's material, organic lesions of the heart were found.

The frequency of cardiac disease in chorea varies as given in hospital and ambulatory statistics. The severer cases come to the hospitals. The majority of the ambulatory cases are mild. Thus 39 per cent. of my hospital cases showed a cardiac lesion (endocarditis), while only 13 per cent. of the ambulatory cases were similarly affected. There would thus be an average of 26 per cent. of both hospital and ambulatory cases. The lesions in simple chorea referable to the endocardium usually affect the mitral valve. Of 17 valvular lesions, 14 occurred at the mitral valve (systolic). The aortic valve was affected in 3 cases (Fig. 138). Pericarditis

FIG. 138.



Chorea. Recurrent attack of moderate severity. Systolic murmur over the aortic area. Fourteen days of the temperature is shown here. Child, twelve years of age.

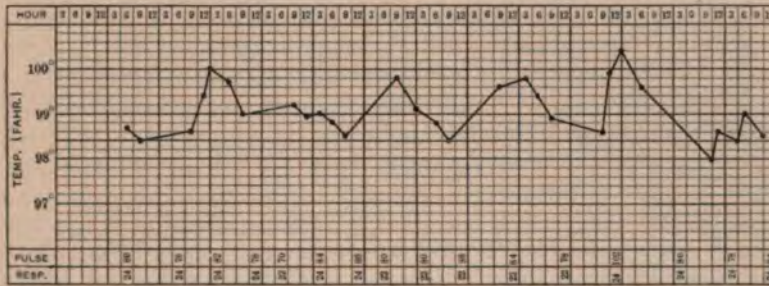
occurred in one of my cases. In the majority of cases in which there was endocarditis either the patient or the parents gave a rheumatic history. On the other hand, not all murmurs of the heart are organic. In 9 per cent. of Starr's 1400 cases, there were functional murmurs heard at the base and over the pulmonic area, early or late in the disease. A gentle blowing at the apex which is heard to the left of the sternum and is not conducted into the axilla or arteries is heard late in the affection, and is undoubtedly hæmic or myocarditic (Osler). I have heard these murmurs in many cases and have come to the same conclusion. Murmurs may also arise at the tricuspid orifice. The organic murmurs are,





twitchings, which so closely resemble habit movements that it is often not easy to come to a conclusion in regard to them. Sachs thinks that the twitchings of the tongue are a means of distinguishing the mild cases of chorea from cases of habit movements. If the patient is told to show the tongue, the tremors and twitchings of that organ and the facial grimaces at once become marked. The movements of the muscles are more rhythmic in hysteria than in chorea. True Sydenham's chorea should be distinguished from the chorea and athetoid movements seen in cases of infantile palsy. The history of the cases, the paralysis, the condition of the reflexes and the contractures will be of assistance in making a diagnosis. True Sydenham's chorea should also be differentiated from cases of tic convulsif and habit movements. A diagnosis of chorea, made in a case which has lasted for a year or more, is open to doubt.

FIG. 140.



Chorea, without endocarditis, two months in duration. No rheumatic history. Female child, nine years of age.

The **duration** of chorea is variable. It may last from three to ten weeks, and may recur. The recurrent attacks are not necessarily any more severe than previous attacks. Fully one-third of the cases in some statistics show two or more attacks. Of Starr's 1400 cases, 365, or 26 per cent., had relapses. One case had nine attacks. Starr thinks relapses less frequent in private practice than in hospitals.

The **prognosis** of chorea minor is very good. Recovery is the rule, but in exceptional cases it may be delayed for fully three months.

The **treatment** of chorea consists at first in giving the patient perfect rest and quiet surroundings. Children are put to bed and kept free from excitement. I do not think it necessary to isolate them, and it is not wise to do so, since they may, under such treatment, become melancholic. An ordinary amount of quiet, such as is prescribed in cardiac cases, is all that is usually necessary. The patient may be allowed to look at picture-books, but not to study or to read. A simple, easily assimilable diet is indicated, milk

and eggs being the chief articles. A warm bath is given daily and the spine sponged with cool water, as some authors recommend. I have not found this necessary in all cases, and would advise it to be omitted if the children strongly object to it. Massage is of great value with anæmic children in whom the circulation is below the average and who have no cardiac disease and no temperature.

**Drugs.**—Fowler's solution is used almost as a routine remedy in these cases. In my experience its curative effects are doubtful. I therefore prefer to give it in small tonic doses, rather than risk the ill effects of large dosage. There are cases in which any attempt to administer it causes vomiting, and which therefore do much better without it. In any case it should be well diluted. In this way larger doses can be given for a greater length of time than would otherwise be possible.

Cases which show recent or old endocarditis or which have articular pains should receive antirheumatic treatment. Alkalies to keep the bowels open, alkaline baths, and sodium salicylate are the remedies in use in these cases.

If there is great restlessness, bromides should be resorted to. It is a very good plan to combine the bromides of sodium, potassium, and ammonium in one mixture. Trional given in grain v (0.3) doses several times daily is a very good remedy in this set of cases, especially if there is wakefulness at night.

If on account of the loss of appetite and general mental depression it is not possible to give any drugs, the children are simply kept quiet and given a nutritious diet. They frequently recover without the help of any drugs. In ordinary cases there is no necessity of using opiates, such as codeine. Antipyrin in grain v (0.3) doses has been recommended. I have not found it better than other remedies. Children who have recovered should be kept quiet for fear of a recurrence of symptoms. This is especially true of cases in which the heart has been the seat of a recent endocarditis.

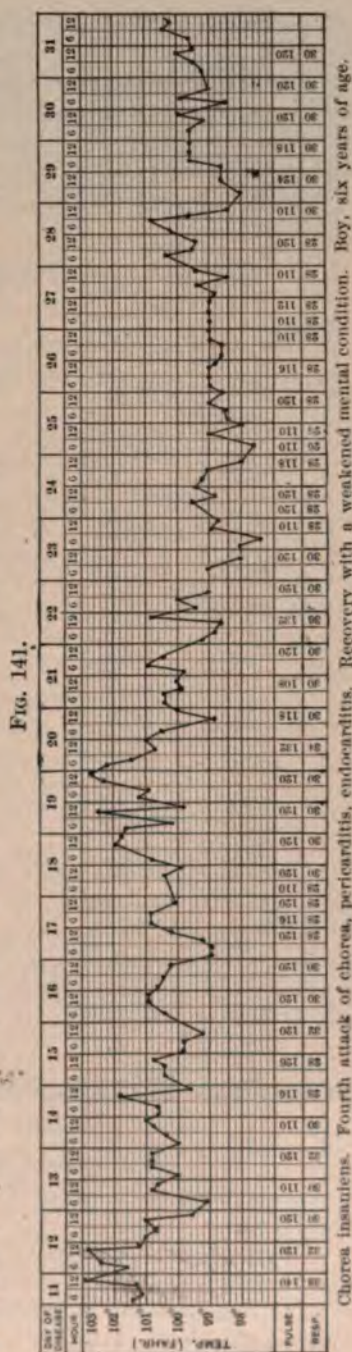
### Chorea Insaniens.

Chorea insaniens is a term applied to the severest form of chorea. A large number of these cases run their course with delirium and high fever. It occurs especially in female subjects. At the outset, there may appear to be nothing more than an ordinarily severe chorea, but the patient rapidly becomes worse. Delirium with hallucinations sets in, finally giving way to incoherency and mania. The patients are in incessant motion, and do not sleep at night. The fever may mount as high as 107° F. (41.6° C.). The cases are in many instances fatal. Osler gives a *résumé* of some fatal cases. I have seen 2 fatal cases of this form. One case occurring during my service as interne at Bellevue Hospital was that of a girl of twelve, who



died with symptoms very similar to those of acute mania. Another case, seen recently, was a boy of ten years, who had for two years previously suffered from ordinary chorea. He had a mitral regurgitant murmur. Two weeks before his death he was suffering from a mild recurrence of the chorea. While in that state he was operated on for adenoids and enlarged tonsils. Chloroform was administered. Three days after the operation the boy was taken with a chill, the chorea became worse, and there was fever. Examination of the heart showed endocarditis and pericarditis with dilatation of the left ventricle. In the second week, the boy became delirious, and did not sleep at night. He complained constantly of pain in the præcordium and tossed in bed. He died two weeks after the onset of the disease. There was throughout a high febrile movement. A third case was that of a boy six years of age, whose temperature-curve is herewith appended (Fig. 141). This case occurred in my hospital service. It was the boy's third attack of chorea. He had chronic cardiac disease. In the final attack there was complicating pericarditis with effusion. The delirium was constant and the choreic movements incessant. He went into a typhoid state, but recovered, his mental faculties, however, being shattered. During the course of the pericarditis there was a polynuclear leucocytosis, and 45 per cent. hæmoglobin.

These cases are to be differentiated from cases of severe simple chorea, in which the movements are so incessant that the patients can with difficulty be kept in bed. In



simple chorea there is no delirium and there is a period of quiescence at night.

The **treatment** of chorea insaniens is symptomatic. The delirium and incessant restlessness are controlled with bromide of potassium, or sodium combined with chloral hydrate. The use of morphine is indicated in cases in which the chloral and bromides are ineffectual. Complicating endocarditis and pericarditis are treated as when primary.

### FORMS OF TIC.

(*Habit Movements or Spasms.*)

This affection is mentioned in this place to emphasize the importance of sharply differentiating its forms from true Sydenham's chorea. Tic is defined by Gowers as a habitual and conscious convulsive movement of one or more of the muscles of the body, reproducing some reflex or automatic movement normal to the individual. Osler has classified the forms of tic. There is first the ordinary form, in which young people or children develop a spasm of a group of muscles, generally of the face. Children do not have the form known as idiopathic spasm of adults in which the lower extremities are involved. There is contraction of a group of facial muscles, such as the orbicularis or the muscles about the nose. There are other forms of tic in which mental disturbances and explosive utterance of words or syllables are prominent features. If the words are of an obscene character, the condition is called *coprolalia*. In other cases the patients repeat words or sentences (*echolalia*). The so-called laryngeal barks of a hysterical nature are, according to most observers, to be classified as forms of tic, and not as laryngeal chorea.

There is a fourth class, which includes those cases in which the subject before proceeding to any definite act, such as writing, feels impelled to blow on the fingers, pinch the nose, or strike the head or thorax. These actions may be regarded as harmless tricks. In another form of tic the patients feel impelled to touch objects, such as the floor or wall (*delire de toucher* of French writers).

### RHYTHMIC MOVEMENTS OF THE HEAD ASSOCIATED WITH NYSTAGMUS.

(*Head-nodding; Spasmus Nutans; Gyrospasm.*)

Nystagmus alone is quite frequently observed in infancy and childhood.

Rhythmic movements of the head associated with nystagmus constitute an uncommon affection.

The derangement is functional and occurs in poorly nourished and rachitic infants whose nerve resistance is diminished. The



majority of cases give a history of some preceding illness, in the course of which the infant has suffered from convulsions. The mothers may be of a nervous temperament. The phenomenon which at once attracts attention is a rhythmic oscillation of the head in a horizontal or vertical direction, or both. On close examination it will also be noticed that the eyes have a horizontal, vertical, or oblique form of nystagmus. Ebert, Cahen, Caillé, Gee, Hadden, and Lewi have studied these cases. Lewi reported 6 cases from my clinic. The ages of the infants ranged from three to eighteen months. The movements were augmented when the infant focussed some attractive object. The nystagmus, if not marked, may be made apparent by holding an object to the right and upward for the infant to focus. Lewi as well as Caillé found that the nystagmus ceased when the infant was blindfolded. In one case the movements continued when the infant was in the recumbent posture. The eye and head movements were not synchronous. As a rule the eye movements were the more rapid. These observers did not agree with Hadden in finding that forcible restraint of the head stopped the nystagmus. I have been accustomed to see a number of these cases yearly. Some of the infants are quite bright and well nourished. This statement agrees with that which Thomson recently made. Three-fourths of the cases are under the age of twelve months (Thomson).

The **etiology** of the affection is obscure. It is usually coincident with the period of dentition, but may appear as early as the third month. Some of the infants live in dark, squalid quarters, and the affection has been attributed to eye-strain caused by the infant's attempts to fix a light as it lies in its crib. This theory would make the affection appear similar to that frequently seen in miners (Magnus). Some of the patients that I have seen lived in well-lighted quarters.

Rachitis was present in most of my cases. Thomson's experience was similar. Henoch gives a physiological explanation of the combination of nystagmus with the rotary movements of the head, by pointing out that the root nuclei of the nerves of the muscles of the neck and throat which rotate the head are adjacent to the ocular nuclei, and that any irritation of one set of nuclei may affect the other. This explanation has been generally accepted.

**Treatment.**—The cases as a rule recover. They are given outdoor air, correct food, and a general course of treatment for the rachitis. Phosphorus is given as in rachitis. I have also prescribed the bromides of potassium and sodium, grains v (0.35) three times daily, but cannot say that they have had curative effects. The cases certainly improved in time. The blindfolding suggested by Caillé only stops the rhythmic movements of the head temporarily.

## HYDROCEPHALUS.

(*Dropsy of the Brain.*)

Hydrocephalus or dropsy of the brain is an abnormal accumulation of fluid in the subdural space, or in the ventricles of the brain. In the former case there is external, in the latter, internal hydrocephalus. Hydrocephalus may be acute or chronic. It may also be congenital, secondary, or primary. The last-named form occurs in adult subjects (Delafield).

### Acute Hydrocephalus.

Acute hydrocephalus, internal or external, is a form of serous meningeal inflammation which may be primary or secondary. Formerly tuberculous meningitis was known as acute hydrocephalus. Forms of acute hydrocephalus, apart from that due to tuberculous meningitis, are secondary to acute disease, such as typhoid fever, pneumonia, and tumor of the brain, or they follow a traumatism or cerebro-spinal meningitis and other forms of meningitis of the non-tuberculous variety.

This form of serous effusion, both when outside and within the ventricles, originates in an obstruction of the venous or lymphatic circulation.

The **symptoms** of the primary form are indefinite. There is fever. Headache, rigidity of the neck, nausea, vomiting, stupor, coma, and delirium are among the initial symptoms. The pupils are sluggish and there may be optic neuritis, convulsions, and paralyses of various kinds, including paralysis of the external rectus of the eye. The symptoms closely resemble those of meningitis. The condition is rare before the end of the first year, and is most common between the first and fifth years.

**Course.**—In mild forms recovery may take place; the severer forms are fatal.

### Chronic Internal Congenital Hydrocephalus.

The accumulation of fluid begins in utero. The quantity at birth may be small and may afterward increase. It may be large enough at birth to obstruct delivery.

**Etiology.**—The causes of the condition are unknown. Alcoholism, syphilis, and tuberculosis of the parents have been regarded as predisposing causes, but infants thus affected may be born of perfectly healthy parents. Sometimes several infants with this malady are born to one mother.

**Morbid Anatomy.**—The quantity of fluid accumulated in the ventricles varies. The fluid is perfectly clear and has a specific gravity



of from 1001 to 1009. It contains a trace of albumin and sometimes urea, sodium chloride, and cholesterin. The weight may reach twenty-seven pounds. The fluid distends the lateral ventricles, the third and fifth ventricles, and the fourth to a less degree. The central canal of the cord may be dilated (Delafield). The corpus callosum is displaced upward. The thickness of the cerebral substance may be reduced to a few millimetres. The convolutions may be obliterated, as may also the basal ganglia. The aqueduct of Sylvius is dilated. The white matter of the brain suffers most. The membrane of that organ may be normal. The ependyma may be thickened and granular.

The **symptoms** are the gradually increasing size of the head and the development of idiocy and paralysis as a result of

FIG. 142.



Congenital internal hydrocephalus. Infant, nine months of age.

internal pressure on the nervous structures. The cranium enlarges so that it becomes disproportionate to the face, which remains small. There is bulging of the occipital and frontal regions. The orbital plates take an oblique direction, causing the eyes to assume a peculiar stare (Fig. 142). The sclera is seen exposed above the cornea. The eyes are directed downward and are only partially covered by the eyelids. The sutures are forced apart and the fontanelles are widely open. The anterior fontanelle bulges and pulsates visibly. The cranial bones may here and there show areas of thinness resembling those seen in craniotabes. The lambdoid suture

is flattened and the greatest diameter is across the temples. The head may attain an enormous size, the child being unable to hold it upright. The hair is scanty and dry. There may be strabismus, palsies, contractures, and convulsions. The eyes may not be on a level. Blindness may result. When the disease is progressive, idiocy develops. The children are very weak.

**Diagnosis.**—Hydrencephaloid or spurious hydrocephalus is a condition which supervenes in acute exhausting states, such as that which follows diarrhœal diseases. There is neither bulging of the fontanelles nor enlargement of the head. The fontanelle is depressed and the eyes are sunken. In certain forms of rachitis which are accompanied by craniotabes and cranial bosses over the parietal and frontal bones, there is frequently a very mild form of hydrocephalus. This condition is rarely progressive. It may be distinguished from true congenital hydrocephalus by the absence of progressive enlargement of the skull. The sutures may be patent, especially that between the parietal and frontal bones. The signs of rachitis are present elsewhere, and the children are, in contrast to the semi-idiotic subjects of hydrocephalus, very bright.

In differentiating internal congenital hydrocephalus from the external form, the history is of great value. External hydrocephalus appears at birth and is not accompanied by bulging of the frontal and occipital bones. Mental deficiency is present from the outset. Late in the disease it may be impossible to distinguish between the two forms. A form of cranial syphilis is mentioned by Gowers as causing cranial enlargement, which, however, is never so marked as in congenital hydrocephalus.

The diagnosis of congenital chronic internal hydrocephalus rests on the progressive enlargement of the cranium, the bulging in the occipital and frontal regions, and the flattening across the lambdoid suture. Acquired hydrocephalus rarely appears before the tenth month (Ireland).

It is sometimes of interest to distinguish at autopsy between the congenital and acquired forms of hydrocephalus. Meynert has shown that in congenital hydrocephalus the lateral ventricles are dilated in their long diameters; the posterior horn is dilated, so that it reaches within a few millimetres of the cranium. Acquired hydrocephalus, on the contrary, usually dilates the ventricles in their vertical and cross diameters.

**Prognosis.**—Hydrocephalus is one of the most fatal nervous affections. There are mild forms in which the accumulation of fluid ceases after a certain time and recovery takes place, the intelligence being either slightly weakened or normal. In some cases the enlargement continues and death ensues from marasmus. In other cases the head becomes of enormous size; the increase of fluid ceases; the fontanelles and sutures close; the unfortunate subjects have an



enormous ossified skull, which they are unable to hold upright. They are partially idiotic or imbecile. They often, however, have a slight degree of intelligence, and may recite lessons, but are helpless in every way.

The **treatment** of congenital internal hydrocephalus is alone of interest to the physician. The condition is hopeless. The injection of solutions of iodine (Morton's fluid) has been tried with doubtful results. I have had 2 cases in which the ventricles were aspirated, fluid was withdrawn, and the head bandaged. The operations were performed by an expert under antiseptic precautions. In neither case was the course of the disease affected. The fluid reaccumulated. Both patients died. On another infant, I performed repeated lumbar puncture at intervals of weeks. The infant seemed brighter after each operation. Several days after the last puncture the temperature rose to  $108^{\circ}$  F. ( $42.2^{\circ}$  C.), Cheyne-Stokes respiration set in, and the patient died.

Cases in which Keen, of Philadelphia, inserted a permanent drain did not give encouraging results. Pott had an equally discouraging experience with that mode of treatment. Iodide of potassium administered internally is of doubtful value. In estimating the results of treatment, it should not be forgotten that a small percentage of cases cease to progress at a certain stage of the disease, and make a tolerably fair spontaneous recovery.

### External Hydrocephalus.

External hydrocephalus may be acquired or congenital. If congenital, it follows an intra-uterine pachymeningitis or may take place because of the rudimentary state of the cerebrum (hydrocephalus anencephalique). External hydrocephalus may be acquired, in which case it follows a pachymeningitis internal hæmorrhagica or is the result of a meningitis in infancy. I have seen such cases. The congenital form of external hydrocephalus is very rare. Bokai records a case in an infant nine months of age. There was an accumulation of fluid between the dura and the pia mater. Both membranes and the falx were thickened, but there were otherwise no signs of inflammation. The infant had spastic symptoms. The diagnostic points in these cases are the uniform enlargement of the head and the bulging, especially in the temporal region. The axes of the eyes remain normal, the condition of those organs differing in that respect from that seen in internal hydrocephalus, in which they are depressed downward. There may be slight exophthalmos. In Lewis Smith's case, the axes of the eyes were normal.

In some cases of external hydrocephalus the head attains an enormous size. The disease cannot then be distinguished from the

chronic internal form. In one of my cases external hydrocephalus followed meningitis. The head was uniformly large, the bulging over the temporal region being marked. The axes of the eyes were normal. The intelligence was low.

In some cases of external hydrocephalus there is a slight internal hydrocephalus.

### AMAUROTIC IDIOCY.

(*Family Idiocy*—SACHS.)

This disease was first described by Warren Tay, an English oculist, in 1881. Among other symptoms, he noticed peculiar changes in the fundus of an infant suffering from the affection. We owe the more extensive study of the affection to the American neurologist Sachs, who described his first case in 1887, not knowing that Tay and Kingdon had previously published theirs. Sachs has collected 27 cases in the literature, his own cases being included in the number. I have published 2 cases and have since seen 6 others.

The **etiology** of the affection is still unknown. Alcoholism and syphilis do not appear to be very closely connected with its occurrence. It appears to run in families. Frequently two or more children in a family are affected.

**Morbid Anatomy.**—Tay-Kingdon, Sachs, and Van Giesen have studied the changes which occur in the nervous system. The cases of Sachs were examined by Van Giesen, who found that the ganglion-cells of the cortex of the cerebrum, and especially the pyramidal cells, showed changes which indicated an arrest of development—an agensis corticalis. There were no changes in the ganglion-cells of the cord and nuclear masses. There were degenerative areas in the white matter of the lateral tracts. Hirsch has lately examined, by new methods, the brain and cord in a case of amaurotic idiocy. He found a uniform degeneration of the ganglion-cells of the gray matter throughout the whole nervous system. There were chromatolysis and displacement of the nuclei of the ganglion-cells toward the periphery of the cell-body, with a destruction and breaking off of the dendrites and axis-cylinders. These changes were found throughout the gray matter of the brain and spinal cord. Hirsch thinks that these changes support his theory of the toxic nature of the disease. He is inclined to regard amaurotic idiocy as a form of infection originating in the intestinal canal.

The **symptoms** are divided as follows: (1) Psychical disturbances tending to complete idiocy. (2) Weakness, resulting after a time in complete paralysis. (3) A normal, diminished, or increased state of the deep reflexes. (4) Increasing blindness with pathognomonic changes in the region of the macula lutea (Tay and Kingdon's spot), with optic neuritis. (5) Marasmus.

The history of all the cases is practically the same. The infant



is normal at birth. After from two to eight months, it is found to be indifferent to its surroundings. It rolls the eyes here and there. Although well nourished, it cannot sit up or hold the head upright. The head falls backward when an attempt is made to cause the infant to sit upright. Many of the infants cry constantly, at the same time making automatic facial grimaces. The lower extremities are weak and may exhibit complete paralysis (diplegia). In other cases, there may at intervals be a spastic rigidity of the lower extremities, alternating with a lax condition. Convulsions are absent or may occur occasionally. The deep reflexes may be normal or diminished. In the spastic cases they are increased. After the first year the infants become totally blind and completely idiotic. They finally become marantic, and die after the second year with the symptoms of advanced infantile atrophy. Occasionally there are nystagmus, strabismus, and hyperacusis. Deafness supervenes in many cases. The electrical contractility of the muscles may be normal or, as in one of my cases, diminished.

**Ocular Changes.**—The changes in the fundus of the eye described by Tay and Kingdon have been confirmed in the cases of Sachs, Koller, Heiman, and in my cases. They are invariably present at some period of the disease, but may only appear late, as in the cases of Koller. Once present, they fix the diagnosis absolutely. The appearances consist of a cherry-red spot on a diffusely white area at the region of the macula lutea. I have seen 8 cases in which they were present. Optic neuritis is also present toward the close of the disease.

**Diagnosis** is not difficult after a study of the symptoms. If an infant is brought to the physician with a history of good health and intelligence up to a certain time, after which weakness and loss of interest in its surroundings set in, with inability to hold the head upright, the fundus of the eye should be examined. If Tay-Kingdon's spot is found, the diagnosis is fixed. I have lately seen a number of cases in which the spastic symptoms were predominant. There were idiocy, increase of reflexes, complete or total blindness, and hyperacusis. I have watched infants with these symptoms for a long time and failed, even with expert aid, to find Tay-Kingdon's spot. In these cases there was probably a birth palsy.

The **prognosis** is invariably fatal. Of the 27 cases of Sachs, only 1 lived to the age of six years. Most of the infants die before the end of the second year.

### TUMORS OF THE BRAIN.

Fully 50 per cent. of the brain tumors in infancy and childhood are tuberculous; gliomata and sarcomata are next in order of fre-

quency. Cysts are secondary to a hemorrhage or embolism. They may remain stationary for a long period, and then increase in size and cause symptoms. Males are affected twice as frequently as females; two-thirds of the cases in male subjects are cases of gliomata and tubercle. Tumors are rare in the first six months of life. The largest number occur in the first decade.

**Location.**—The medulla is rarely the seat of tumor. The cerebellum is most frequently involved (50 per cent. of the cases, Gerhardt, Peterson). The pars centrum ovale and basal ganglia are the parts next most frequently affected.

**Etiology.**—The rôle of traumatism is not clearly understood. Gliomata are due to a proliferation of the neuroglia. Tubercle and sarcoma are secondary to foci elsewhere. Carcinoma is rare. In some cases of that growth the orbit is a focus of infection.

**Symptoms**—Symptoms of pressure and irritation vary with the location of the tumor. A small but rapidly growing tumor will cause more pronounced symptoms than a large tumor of slow growth. Interference with the blood-supply and an increase in the quantity of fluid within the ventricles of the brain will cause the symptoms to vary.

**General Symptoms.**—**HEADACHE.**—This may in cortical and meningeal tumors be intense. It is of a boring, gnawing character, and is referred to the region of the tumor. Tumors in infants may attain great size previous to ossification of the skull. The bones of the skull are pushed apart and the sutures opened up. There is very little pain. Sleeplessness and restlessness, emaciation, and cerebral excitement are marked.

**NAUSEA and VOMITING** are prominent symptoms and persist for a long time. The vomiting is projectile and occurs independently of the ingestion of food.

**VERTIGO** is common and occurs with every change in the position of the head. It is a common symptom in tumors of the pons and cerebellum.

**CONVULSIONS.**—These may be localized or general. They occur when the cortex and motor areas are invaded, and eventuate in epilepsy of the Jacksonian type. In this form of epilepsy, the attack begins in the head or arm corresponding to the area of irritation, and subsequently becomes general.

**OPTIC NEURITIS** and optic atrophy are important symptoms of intracranial tumor, but are not always present. When tumors are situated at the base of the brain, the symptoms appear early and are due to pressure on the chiasm. Optic neuritis is either double or more pronounced in one eye.

The **PULSE** and **RESPIRATION** present no characteristic features. They show irregularities in rate. Respiration is affected only toward the close of the affection.



**Symptoms Dependent on the Location of the Tumor.**—**CORTICAL TUMORS** in or near the motor areas cause convulsive seizures, which occur from the outset. Subcortical tumors will at first cause paralysis and, as they encroach upon the cortex, convulsions. With invasion of the cortex there are, in addition to convulsions with subsequent epilepsy, intense headaches. Tubercle, glioma, and gumma occur near the surface. Cysts and sarcoma are more deeply situated.

**THE FRONTAL LOBE.**—The tumors situated in this region cause stupidity and other marked changes in the degree of intelligence. There will be a perversion of the sense of smell, salivation, and also the drooling seen in idiocy. If the third frontal convolution is affected, there will be motor aphasia associated with agraphia—a rare condition in childhood. Tumors of the motor area will in the earlier stages cause cortical irritation, manifested in convulsive twitchings in the parts first paralyzed. There may be slight sensory or motor disturbances in an upper extremity and an occasional twitching of the arm, forearm, or thumb.

The tumors of the **PARIETAL LOBE** cause sensory changes in the limbs of the opposite side of the body (Dana). If the white substance is the seat of tumor, there may be hemianopsia; Wernicke's centre for conjugate movement of the eyes may be affected if the tumor is situated in the inferior part of the parietal lobe.

Tumors of the **OCCIPITAL LOBE** cause homonymous hemianopsia with or without epileptiform convulsions, the latter being probably due to invasion of the cortex.

Tumors of the **TEMPOROSPHENOIDAL LOBE** cause impairment of hearing on the side opposite to the lesion and sensory aphasia. The patient is able to speak, but cannot understand what is said or repeat spoken language.

In tumors of the **GANGLIA**, there is involvement of the internal capsules. There are no convulsions and none of the choreic and athetoid movement seen in cortical tumors.

Tumors of the **CRUS CEREBRI** cause paralysis of motion and sensation on the opposite side of the body, and oculomotor paralysis, ptosis, and paralysis of the muscles of the eyeball, except the external rectus and superior oblique. There will be paralysis of the sphincter iridis and ciliary muscle. There may be paralysis of both sides of the body, double ptosis, and double oculomotor symptoms. The majority of cases are at first unilateral, later becoming bilateral. Loss of pupillary reflex, nystagmus, and cerebellar ataxia point to involvement of the corpora quadrigemina.

Tumors of the **PONS** cause unilateral or bilateral symptoms. There is hemiplegia or double hemiplegia with paralysis of the cranial nerves. There is paralysis of the third, fifth, sixth, seventh, and eighth nerves of the side of the lesion, with hemiplegia of

the opposite side. There may thus be paralysis of the external rectus with facial palsy and impairment of hearing on one side. If the nucleus of the sixth nerve is involved, there will be paralysis of conjugate movement of the eyes toward the side of the lesion, while if it is not affected there will be only external rectus palsy of the side of the lesion not affecting conjugate movement of the other eye.

Tumors of the MEDULLA manifest themselves in bulbar symptoms. There will be paralysis of the glossopharyngeal, vagus, spinal, accessory, and hypoglossal nerves. Thus there are unilateral or bilateral paralysis of the arms or legs, difficult deglutition, and disturbances of the respiratory movements and of cardiac action. In addition there will be spasm of the sternomastoid and trapezius muscles, and paralysis of the tongue, with atrophy, vomiting, polyuria, and glycosuria; optic neuritis occurs early, and there is severe occipital headache. Gummata in this region are not uncommon.

Tumors of the CEREBELLUM, which are usually of the solitary tuberculous form, are the most important intracranial growths in children. There will be occipital headache, vomiting early in the disease, and cerebellar titubation due to encroachment upon the middle peduncle. Vertigo is severe. The sixth, seventh, or eighth cranial nerves may be involved. There may be bulbar symptoms. Paralysis of the external rectus is very common in these tumors. Optic neuritis may be present.

This list is by no means complete. The notes are given as concisely as possible. For further details the student is referred to the extensive monographs on Tumors of the Brain.

### INFANTILE CEREBRAL PALSY.

(*Spastic Hemiplegia; Diplegia; Paraplegia.*)

**Forms.**—All clinicians of note now classify these palsies with regard to the time of onset. There are three varieties—the intra-uterine or prenatal, the birth palsies, and the post-natal forms.

Brain palsy is a common disease of infancy and childhood, and has been known to occur up to the tenth year. Gowers and Osler are agreed that these palsies are most frequent in the first two years of life. They occur with equal frequency during the first and second years.

The **etiology** of the affection is still very obscure, and differs in the various forms. In the intra-uterine or prenatal form, the causal influences are especially indefinite. The influence of maternal impressions, such as fright or worry, is uncertain. Other causes frequently cited are hereditary insanity or neurotic affections, an injury or blow to the abdomen, exhausting fevers during pregnancy, pneumonia, typhoid fever, and uræmic convulsions. The rôle played in



this disease by syphilis is as yet undetermined. That of alcoholism is also obscure.

The **birth palsies** have been studied by Little and McNutt. To the latter we owe the first lucid post-mortem demonstration of their cause. She published several cases under the title of *Apoplexia Neonatorum*. It was demonstrated in this article that in easy labors as well as in prolonged and instrumental deliveries, hemorrhages on the surface of the brain occurred and were the cause of subsequent palsies, with the resulting contractures and idiocy seen in such cases.

**The Post-natal Cases.**—The etiology of these cases is still a matter of discussion. When Strümpel proposed the theory of an encephalitis similar to that occurring in infantile poliomyelitis, it was for a short time accepted. Clinically this theory was founded on certain similarities between the spinal and cerebral affections. It has since been abandoned. It is found that many of the cases follow the acute infectious diseases, especially measles and scarlet fever (Gowers). Cerebral palsy may follow typhoid fever, pertussis, pneumonia, amygdalitis, cerebrospinal meningitis, gastro-enteritis, and traumatism to the skull. Infection or the presence of infectious disease cannot alone explain all the cases. The view most generally accepted is that the convulsion at the outset of the disease causes the bursting of a vessel weakened by some form of degeneration (Osler).

**Morbid Anatomy.—Prenatal Cases.**—There is porencephaly. Half a hemisphere, an entire hemisphere, or both hemispheres may be imperfectly developed. There are also certain defects in the cerebral hemisphere to which is applied the term "*Agensis Corticalis*." That is to say, there is imperfect development of the cortical gray cells, particularly those of the pyramidal type. The agensis may extend throughout all parts of the hemispheres.

**Birth Palsies.**—The principal lesion is meningeal hemorrhage (McNutt). This may occur in areas over the cortex, or at the base of the brain. There may be a diffuse hemorrhage over the whole cortex of one hemisphere. The extravasation is, as a rule, most profuse over the motor area.

**Acute Palsies.**—In these, there are found embolism and thrombosis, or hemorrhage, the latter occurring mostly at an advanced age. As a result there may be atrophy of the cortex, sclerosis or cyst formations. Cysts are sometimes found later in life, there having been no previous symptoms (Gowers). They undoubtedly originate in infancy. Some authors (Gowers) state that embolism, others that hemorrhage, is the pathological condition most frequently found in cerebral palsies of acute origin. The cause of hemorrhage in these cases is still a matter of speculation. There is certainly a change in the bloodvessels, but whether it is the fatty change seen

in the bloodvessels in infancy and first pointed out by von Recklinghausen, is a question. It may be that, given a vulnerable bloodvessel, heart disease or any infectious disease will predispose to hemorrhage. Cysts are likely to be found in cases in which there is idiocy.

**Symptoms.**—In the prenatal cases the infant is born with the disease partially or completely developed. There are cases in which no symptoms are seen at birth. They develop during the first year.

**Birth Palsies.**—In these cases, as in those of McNutt, there are symptoms of hemorrhage, disturbance of respiration, partial or complete loss of consciousness, and convulsions which may be general, or involve only one side of the body. The majority of infants thus affected die soon after birth; others live to exhibit various symptoms of defective development of the brain. There are paralyzes more or less complete, with contractures, spastic rigidity of sets of muscles or of all the muscles, and increase of tendon reflex.

The clinical history of the very mild forms of cerebral birth palsy is characteristic. Many of the cases are mistaken for cases of asphyxia neonatorum. After a normal delivery, the infant is noticed not to breathe deeply or regularly and to appear at times to cease breathing. If roused, it cries feebly, but again relapses into a quiescent state, the breathing being irregular and shallow. It becomes slightly cyanosed, and while in this state may have repeated convulsions. In other cases the infant is born apparently well, but after twenty-four hours the respirations become shallow and increase in frequency, and convulsions appear. These cases may recover. At about the time when the infants attempt to walk they may show slight spastic symptoms in the lower extremities, and have overextension of the foot and toes as soon as any attempt is made to place the feet on the ground.

In severe cases unilateral paralysis and weakness of the muscles which support the head remain permanent. Spastic and paralytic symptoms are in some cases very marked. Athetoid movements of the extremities, with inability to grasp objects, contractures of the lower extremities, adduction of the thighs, mental defects, inability to sit upright, and oscillation in walking complete the later picture.

**Acute Cerebral Palsy.**—According to Gowers, the onset is acute in two-thirds of the cases occurring before the end of the second year. The condition occurs with the same frequency in the first and second years, and is as a rule primary (Gowers). Although, as has been stated, it may follow the infectious diseases, there are frequently no premonitory symptoms; the patient retires in good health and awakens with a hemiplegia. In more than half the cases the disease



is ushered in with convulsions, generally unilateral. Whether these are present or not, there is unconsciousness lasting a few hours or for days, and sometimes fever and vomiting. When the child recovers the hemiplegia may be complete or the paralysis may develop slowly. The right side is more often involved (Osler).

The symptoms in order of occurrence are as follows :

*Convulsions.*—Coma and convulsions may be present at the outset, but may not develop until later in the disease. They are most likely to occur at the outset of the infectious diseases. If they persist into the later periods, epilepsy is likely to develop. I have seen a case in which as many as forty convulsions of an epileptiform character occurred daily. A condition of weak-mindedness or complete imbecility supervenes. Many of these cases are mistaken for true epilepsy.

*Paralysis.*—In all forms of cerebral palsy, there may be hemiplegia, diplegia, or paraplegia. As a rule, hemiplegia is of the right side (Osler). The facial muscles of the same side may be involved. There may be no facial paralysis. Monoplegia, especially of the leg, is rare. Diplegia is not common. After a time, contractures of the extremities occur. With these changes, there are characteristic disturbances of motion. There is the gait of the hemiplegic, slightly or markedly developed. As has previously been stated, the children may be born with contractures. A spastic paraplegia with athetosis in the upper extremities indicates the possibility that the arm may have been involved earlier in the disease. In about 20 per cent. of the cases, the face is involved at the outset. Motor aphasia is present. It is not present in birth palsies. According to Freund, hemianopsia is occasionally present.

*Disturbances of sensation* are rare.

*Reflexes.*—The deep reflexes are increased. There is no atrophy of the muscles as in infantile spinal palsies, or it is at most slight.

With the *ataxia*, there are the athetoid movements first described by Hammond. These are sometimes marked. In some cases, there are cataleptic phenomena. In all cerebral palsies of whatever origin, paralysis, rigidity of muscles, contractures, and increase of the deep reflexes are constant features.

*Diagnosis.*—Intra-uterine and birth palsies give a distinct history of early development. If a palsy has developed a few months after a normal labor, it is to be classed as possibly intra-uterine. Both prenatal and birth palsies are likely to be diplegic or paraplegic. As a rule there is mental deficiency. Paralysis may be complete, or, as in one of my cases, scarcely noticeable. Double athetosis is indicative of double hemiplegia, and may even take the place of paralysis. Choreiform movements are frequently mistaken for chorea. They are unilateral and combined with exaggerated reflexes and partial, slight or marked paralysis. Aphasia of cerebral

palsies is motor rather than sensory. Its presence precludes the possibility of the palsy's being of prenatal or of birth origin.

The cerebral palsies are differentiated from the infantile forms of paralysis by the presence of contractures, rigidity, increase of deep reflexes, and occasionally by the presence of athetosis and choreiform movements. In recent cases the absence of atrophy will also aid in diagnosis.

**Prognosis.**—So far as prenatal and birth forms of palsy are concerned, no definite prediction in regard to the outcome can at first be made. Many of the cases of birth palsy die at the outset. Some escape with very slight paralysis. Others develop convulsions with subsequent epilepsy and idiocy. Contractures, diplegia, and double hemiplegia with spastic symptoms may develop. The acute cerebral forms may improve to such an extent that only slight paralysis, choreiform movements, or athetosis remain. In other cases improvement is followed by a return of the symptoms, with convulsions and epilepsy. It is estimated that fully 45 per cent. of the cerebral palsies develop epilepsy, while the diplegic forms are less likely to do so. One convulsion is apt to be followed by others, and these in time by epilepsy and mental deficiencies.

The **treatment** of cerebral palsy is ultra-conservative. Cases of birth palsy have difficulty in deglutition. Aid in keeping up the nutrition of the patient may be given by spoon-feeding or feeding with stomach-tubes (gavage). If there are convulsions, bromides in moderate doses are administered. The infant should be kept perfectly quiet. In the acute cerebral cases, if hemorrhage is suspected rest and the application of an ice-bag to the head are indicated. Subsequent convulsions are treated with bromides. The bowels are kept open with calomel. In cases in which there is slightly marked paralysis, massage and the various forms of hydrotherapy are of great utility. The faradic current has much the same effect as massage. If contractures and choreiform movements supervene, the various orthopædic appliances are of great practical utility. Where indicated, they should be used in connection with judicious tenotomy. Surgical interference has been practised in forms of epilepsy which simulate the Jacksonian type. The results are disastrous in young children, nor is permanent relief to be expected in older ones.

## FACIAL PALSY.

(*Bell's Paralysis.*)

Paralysis of the facial nerve is quite common in infancy and childhood. As in the adult, the distribution and etiology of the paralysis vary.

The facial paralysis observed in infants who have been delivered



with forceps is a pressure paralysis. It may affect the upper or lower branches of distribution. The prognosis of this form of paral-

FIG. 143.



Facial paralysis, left side, rheumatic form. Girl, eight years of age.

FIG. 144.



Facial paralysis, rheumatic form, showing inability to close the eye. Girl, eight years of age.

ysis is, as a rule, very good. Recovery takes place after a few weeks. Some cases do not thus recover; there should therefore

be some conservatism in prognosis. Congenital facial palsy may occur in the absence of any history of traumatism or pressure. Hensch records such a case in a boy of ten years. There was deafness on the side of the paralysis, but no history of disease of the ear.

The so-called rheumatic form of facial paralysis occurs in infants and children, but rarely does so before the third year, and most commonly between the sixth and fifteenth years. The symptoms are the same as in later life (Figs. 143 and 144).

Of greatest interest to the practitioner are the facial palsies which occur in infants and children as a result of ear disease or of inflammatory disease of the mastoid process. In infants a few months old, I have seen facial palsy due to otitis in one ear (Fig. 145). Hensch has seen cases in infants from three to five

FIG. 145.



Facial palsy complicating otitis. Infant, seven months of age.

months of age. The facial nerve is affected as it passes through the Fallopian canal. Caries of the bone, pus, or swelling in the vicinity of the canal, will cause this form of paralysis. It is therefore a species of pressure paralysis. There may be no distinct collection of pus in the mastoid cells, but, when opened up, the mastoid is found to be filled with granulations. Temperature, tenderness, and redness over the mastoid should arouse suspicion.

Bokai reports a case of retropharyngeal abscess in which the facial palsy was caused by pressure on the nerve as it emerged from the stylo-mastoid foramen.

Another form of facial palsy is that seen in basilar disease of the brain. The facial palsy seen in tuberculous meningitis and sometimes in the non-tuberculous variety is of great diagnostic



import. This paralysis is not always marked; it is often a very slight paresis with flattening of the facial muscles on one side and accompanied by slight widening of the palpebral fissure on the same side. In connection with this symptom, a dilatation of one pupil or slight strabismus is exceedingly significant of basilar affection. In other words, in the forms of meningitic facial palsy, the physician should be on the alert for changes in the contour of the face, since in many of these cases the patient is conscious only at intervals. In many cases, restlessness on the part of the patient will cause the slight flatness of the face or widening of the palpebral fissure to disappear. The patient should be watched unawares or when at perfect rest. The facial palsies with cerebellar tumors and tumors of the pons have been referred to in the section on Tumors.

Operative facial palsy in infants and children is likely to occur after the radical operation on the mastoid, if the operator is not a thorough anatomist. I have felt that this accident could be avoided. After an operation on the mastoid I have seen mild facial palsy, consisting of a very slight lagophthalmos with slight flattening of the facial muscles, which disappeared within twenty-four hours. It was possibly due to pressure on the nerve during the operation. Facial palsy following a mastoid operation is, as a rule, due to actual traumatism to the nerve, and to its partial or total destruction. The paralysis in such cases is permanent.

The **treatment** of facial palsy in infants and children is determined by the origin of the palsy, and is essentially the same as in the adult.

### MULTIPLE NEURITIS.

This is an affection in which several or most of the peripheral nerves undergo degeneration of an acute type. The nerves affected are, as a rule, symmetrically distributed.

**Etiology.**—The disease may be caused by the poisonous action of drugs, such as lead, arsenic, and alcohol. It follows the infectious diseases—measles, diphtheria, typhoid fever, influenza, and malaria. In such cases the degeneration is due to the action of bacterial toxins on the peripheral nerves. Cold is said to favor the onset of the disease. In many cases, it is impossible to fix upon any definite cause.

**Morbid Anatomy.**—There is an early stage during which there are hyperæmia and swelling of the sheaths of the nerves, which may be the seat of minute hemorrhages. The nuclei of the sheaths are enlarged. There is an increase of connective-tissue cells between the nerve-sheaths, and also of round and spindle-shaped cells between the nerve-fibres. The changes in the nerve-fibres are characteristic of nerve degeneration. The muscles may be the seat of parenchy-

matous degeneration. The striation may become indistinct. In some cases there are also interstitial changes.

The **symptoms** of multiple neuritis in children are very characteristic. After an infectious disease, the child no longer walks with a steady gait, but may stumble and fall. After a time it is noticed that the patient does not care to stand, and the mother is unable to persuade it to do so. The child cries when put on its feet, which refuse to support it. There seems to be pain connected with an attempt to stand, and also on handling and pressing the muscles. After a time the child does not sit upright, but falls back or toward one side when put in the sitting posture. It finally becomes completely paralyzed. The paralysis is progressive and symmetrical. The child does not use the hands. The feet drop forward (foot-drop) and there is a very characteristic wrist-drop. The child lies helpless in the crib, unable to move. Some of these patients cry constantly as if in pain. During this time there is good nutrition and the appetite is good. The muscles of the trunk are frequently affected as well as those of the extremities. In these cases there is a species of paralytic lordosis when the child stands or sits upright. In a few cases the muscles of the eye are affected, and in fatal cases those of the diaphragm.

The facial and hypoglossal nerves are rarely the seat of the disease. The musculospiral and peroneal nerves seem, as in poliomyelitis, to be affected. The reflexes are diminished and finally disappear. The dorsum of the feet and hands is slightly affected with oedema.

**Sensory Disturbances.**—In spite of statements to the contrary, it is very difficult in children and infants to elicit exact data as to the pain or sensory changes and their distribution. I have found evidences of pain on handling the children or attempting to make them stand or sit. The patients are restless at night, and cry most of the time, and it must therefore be inferred that they have pain.

**Course.**—The majority of the cases make a complete or almost complete recovery. In a case which I watched very closely the reflexes were slow to return, although the child began to sit upright, then to stand, and finally to walk. The gait in walking was very peculiar. It was a sort of waddle, resembling that exhibited in congenital luxation of the hips. The boy, three years of age, finally made a complete recovery.

As a rule, the symptoms increase in severity for from four to six weeks; they then retrograde and improvement sets in. In some cases the development of symptoms is rapid, the diaphragm becomes affected, and the children die of bronchopneumonia. If the vagus is affected, death occurs through cardiac failure.

**Diagnosis.**—If the clinical picture is studied, the diagnosis is



not difficult. The complete and absolute paralysis is, in its mode of onset and its symmetrical distribution with anatomical impairment of sensation of all kinds, so peculiar that it cannot be confounded with poliomyelitis. In the cases which I have seen, the muscular atrophy was also less marked than in the latter disease. The very characteristic feature of the paralysis is its flaccidity. If the child is

FIG. 146.



Multiple neuritis in a child two and one-half years of age. Shows the complete relaxation of the glutei muscles. Recovery.

made to sit upright, the glutei muscles flare, as it were, outside the body-line and do not retain the tonicity of the normal muscle. There is nevertheless not much atrophy of the glutei. Landry's paralysis is so rare in infancy and childhood that it need not be considered in detail.

The **treatment** is palliative, since the disease is not only self-

limited, but also tends to spontaneous recovery. The pain is relieved and the skin kept in good condition by massage. If the child is restless, it is treated in the ordinary way. There is no specific for the affection. Electricity is not recommended by those whose experience gives weight to an opinion. If contractures result, orthopedic appliances are indicated as in other paralytic diseases.

### ERB'S PALSY.

(*Obstetrical Palsy.*)

This form of palsy, which occurs in infants and children as well as in adults, is due to a neuritis caused by direct traumatism either to the nerves supplying the muscles of the shoulder, or as in the newly born infant by traction or pressure on the brachial plexus (see Fig. 27). Erb showed that the point injured in these cases is the spot between the *scaleni* at the exit of the fifth and sixth cervical nerve roots. Duchenne, Seeligmüller, and Henoch have described these birth cases in infants. I have seen cases in older children which correspond to the adult cases.

The **symptoms** are very characteristic. There is complete paralysis of the arm on the affected side. The child, if directed to raise the arm or forearm, is unable to do so. The fingers can be moved. Infants sometimes hold the paralyzed arm with the healthy one. In a few cases there seems to be pain, caused by the drag of the paralyzed member on the shoulder. After a time there is atrophy of the *deltoid* and other muscles about the shoulder-joint, which causes the bony prominences to show markedly (Plate XXI.). The atrophy sometimes comes on very rapidly. In infants and children it is impossible to reach any conclusion in regard to the intensity of pain and the disturbances of sensation.

The cases should be differentiated from cerebral birth palsies. Apart from the electrical reaction, the absence of hemiplegia or diplegia of a spastic nature with rigidity, the absence of increased reflex, and also of convulsions, all of which are present in birth palsies, will aid in the diagnosis. Later in life it may not be possible to determine which form is present.

The **prognosis** is good, but I have seen severe cases of obstetrical palsy which failed to recover.

The **treatment** depends on the origin of the palsy. If it is obstetrical, the arm should be put in an apparatus to protect it from injury. After two weeks, friction, massage, and a mild electrical current of the faradic variety should be applied. If contractures develop later, splints should be constructed to counteract the tendency. On the whole, the management of the cases is based on the principles which govern the treatment of peripheral palsies.



PLATE XXI.



Erb's Paralysis in a child twenty-six months of age. Atrophy of the deltoid, subluxation of the arm; bony prominences marked.

1

2



**HEREDITARY ATAXIA.**

(*Friedreich's Disease; Hereditary Ataxic Paraplegia.*)

This is a form of ataxy which frequently affects several members of the same family. Rüttimeyer and Griffith collected 233 cases which were distributed in 107 families. In 38 cases there was a direct hereditary history. In the remainder, there was a history of alcoholism, syphilis, or consanguineous marriage. Sixty-five cases of Gowers were distributed among 19 families. Thus there was an average of 3 to each family. In some families there were 10 cases. Isolated cases are rare, and occur, as a rule, only in children. The disease affects the sexes equally. Cases have occurred as early as the second year, and as late as the twenty-fourth, but are seen most frequently between the seventh and eighth years.

**Symptoms.**—The onset of the disease may be gradual or abrupt. The first symptom is an impairment of coördination in the lower extremities. The patient is unsteady in walking, and stands with the feet wide apart. Some patients reel when the eyes are closed more than at other times. In other cases Romberg's symptom is absent. The feet show the peculiar deformity of pes cavus. The instep is high and the toes overextended. The movements of the arms next become ataxic. The speech becomes slow and halting. Jerking, nodding movements of the head set in. Irritability of muscle is absent from the beginning. The deep reflexes may be present at first, but finally disappear as in true tabes.

Nystagmus is usually present, and may be a very early symptom, appearing simultaneously with the ataxic symptoms. The symptoms connected with the speech may come on very late in the disease.

Optic atrophy is never present, and the Argyll-Robertson pupil of tabes is absent.

Sensory disturbances, such as shooting pains, are rare, but may occur. There is no tendency to trophic joint-affection as in tabes. The sphincters are normal.

Muscular power, although normal at first, diminishes as the disease progresses. There is atrophy of muscle. Spinal curvatures, talipes equinus, and equinovarus result. The loss of muscular power is sometimes limited to the lower extremities.

The mental condition is generally affected. The children are slow at school. Imbecility has been recorded (Gowers).

**Course.**—Once inaugurated, the disease is progressive, but it may remain stationary at any stage for some years. The duration is extended over years. Gowers gives the period as ten to twelve years. The patients finally become bedridden, and, as a rule, die from intercurrent disease. The anatomical changes have not as yet been completely classified. This is due to the fact that in certain forms of hereditary ataxia resembling Friedreich's disease,

Marie and Hoffmann have described changes other than those found in typical cases of that affection. The changes in Friedreich's disease consist in a diminution in the transverse diameter of the cord and a sclerosis of the posterior and lateral columns, involving the pyramidal tracts. The neuroglia and vessels of the tracts are involved; whether this is due to an arrest of development of a congenital nature has not been determined.

**Differential Diagnosis.**—The disease should be differentiated from true tabes. In the latter, there are the Argyll-Robertson pupil and optic neuritis, the visceral crises and shooting pains, but neither head-nodding nor nystagmus. The lack of intelligence and the family history are characteristic of Friedreich's disease.

**Prognosis and Treatment.**—There is no cure for the affection. The treatment is designed to relieve the symptoms.

### ACUTE ATROPHIC PARALYSIS.

(*Acute Anterior Poliomyelitis; Infantile Paralysis; Essential Paralysis of Children.*)

This is the most common form of paralysis in infants and children. It is a disease characterized by loss of voluntary power, taking place within a few hours or days. Some of the paralyzed muscles recover; others undergo atrophy.

**Occurrence.**—There is doubt as to whether the disease occurs in foetal life. Duchenne has reported a case at the twelfth day. Such early cases are apt to be cases of hemorrhage rather than of poliomyelitis. The majority of cases occurring during the first year develop after the sixth month, and three-fifths of the cases before the tenth year are found to occur during the first three years. It affects the sexes equally.

The **etiology** of the disease is still uncertain. Cases occasionally occur after exposure to cold, and after a traumatism or a psychological disturbance, such as fright. The relationship between these predisposing elements and the disease is probably no more intimate than is the case in other affections.

This disease is very frequent in the period of dentition, but since dentition has been regarded as a predisposing cause in most diseases, no close relationship is evident. It has been shown by Medin, Strümpel, and Zuppert that many of the cases of poliomyelitis occur after or during the course of the infectious diseases, especially measles, scarlet fever, and typhoid fever. It is probable that some toxin acting through the blood and blood-vessels causes the degeneration in the cord characteristic of the disease. It is also probable that the epidemic occurrence of the affection is due to its infectious nature. Medin, Pasteur, Sinkler, Putnam, Chapin, and Zuppert have reported epidemics.



**Symptoms.**—There are four distinct periods in the development of the symptomatology: The period of onset, in which the symptoms resemble those of an infectious disease; the period of paralysis; the period of retrogression, in which some of the paralyzed parts recover while others remain permanently paralyzed; and finally, the chronic state, in which there are permanent paralysis and atrophy.

**The Onset.**—This is always acute. In most cases there are fever, vomiting, and diarrhoea. The fever may be slight ( $100^{\circ}\text{F.}$ ,  $37.7^{\circ}\text{C.}$ ) or may mount to  $104^{\circ}\text{F.}$  ( $40^{\circ}\text{C.}$ ).

Complete paralysis sets in after these symptoms have continued for a few hours or days. In other cases the paralysis first attracts attention, and is followed by fever and constitutional disturbances persisting for days. General convulsions may usher in the disease, and be followed by coma lasting for days. At the termination of the coma, the patient is found to be paralyzed. The onset sometimes resembles that of cerebrospinal meningitis. There are headache, vomiting, fever, and rigidity, the paralysis becoming apparent after the subsidence of these symptoms. Other cases have absolutely no premonitory symptoms. The patient goes to bed in health, but in the morning is found to be paralyzed. Patients sometimes suddenly fall, and on being raised up are found to be paralyzed. These are probably cases of acute spinal hemorrhage. In older children pain in the course of the nerves may usher in the paralysis. Pains in the joints and back may succeed the paralysis. Such cases closely resemble those of peripheral neuritis.

**The Paralysis.**—The paralysis consists of a loss of power, which is complete in two or three limbs or in parts of extremities. Seeligmüller found the relative frequency of involvement to be as follows: the right lower extremity, the left lower extremity, the right upper extremity, and the left upper extremity, in the order named. All four limbs may be involved, or only a hand and a leg. If all four limbs are at first involved, there is weakness of the back. The patient cannot sit upright or hold the head erect. The cranial nerves escape, except in very rare cases, in which degeneration or inflammation involves the medulla and its nuclei. There may be symptoms which simulate those of bulbar paralysis.

Different sets of muscles may be involved. After the first onset of the paralysis, some of the muscles may recover. Thus a child who has been unable to sit up or move the arms will recover the power to do so. In such cases one leg only may remain permanently paralyzed.

Paralysis may develop slowly in the course of one or two weeks. After that time it comes to a standstill. In a period of from one to three months either recovery will take place or the paralysis will be complete with accompanying atrophy.

**Atrophy** in the paralyzed muscle is very characteristic of the disease. It may be seen as early as the first week. Accompanying it, and appearing from the fifth to the seventh day, is the reaction of degeneration in the paralyzed muscle and nerve. The faradic and galvanic irritability of nerve and muscle are increased for the first two days. They then rapidly diminish, the former disappearing completely. The galvanic irritability remains increased for from two to six months; it then diminishes, and if the paralysis is permanent, disappears at the end of one or two years. In rare cases all electrical irritability disappears from the onset. In others the faradic irritability in certain fibres and muscles returns after from six to twelve months. These muscles may partially recover, but remain atrophied and weak. There is usually no loss of sensation, but if it does occur, there is incontinence of urine. Reflex at the patellar tendon is lost and myotonic irritability is either lost or diminished. In cervical disease of the cord, or when only the posterior tibial muscle, or the muscles of the feet are paralyzed, the tendon reflex at the knee is present. In rare cases, the inflammation may spread from the anterior horns to the lateral columns. The lower extremities may then be paralyzed but not atrophied, and clonus may be present.

Growth of bone is retarded, and one foot may after a time become shorter than the other. The joints become the seat of subluxations through the laxity of the muscle and lack of support. The articular ends of the bones are not held in apposition. Through the shortening of some muscles and the traction of others there will result various forms of talipes. The muscles in front of the tibia are affected more than those of the calf. The extensors of the thigh are more frequently paralyzed than the flexors.

The muscles of the whole arm may be paralyzed, or, as in Erb's paralysis, only those of the deltoid group. The serratus, the pectoralis, the muscles of the back and neck, and the diaphragm may all be affected.

**Course.**—The mildest cases rarely make a complete recovery. Death is very uncommon and occurs only in the early stages. It may supervene within two weeks from general paralysis or cerebral disturbance. Relapses are rare, second attacks unknown.

**Sequelæ.**—A cord which has once been the seat of this disease is naturally susceptible. Gowers states that he has seen chronic disease of the cord supervene later in life. Progressive muscular atrophy or lateral sclerosis may at some later time appear in the cord.

The **prognosis** of acute atrophic paralysis is good as to life. As to the outcome of the paralysis, a prediction can be made only when all the muscles which show faradic irritability have recovered. Some children who in the second stage have shown complete paresis or paralysis from the cervical region down, gradually regain power in all of the affected muscles, only one limb or part of a limb being



permanently affected. As a rule those parts, which after a week respond to faradism, will recover.

**Diagnosis.**—At the onset, the case should be distinguished from one of the infectious diseases. Since the mode of onset is much the same, it is best, as in those diseases, to defer making the diagnosis until the initial symptoms have passed and the paralysis appears. When the paralysis is fully developed, it should be differentiated from forms of cerebral palsy. This in the majority of cases is difficult. The characteristic atrophy, the complete paralysis, the loss of knee-jerk, and the absence of contractures will all be of service. Those cases in which pain in the course of the nerves is present at the onset, should be distinguished from cases of multiple peripheral neuritis. Time and study of the cases will make this possible. In those forms of poliomyelitis in young infants, in which the muscles of the deltoid group are affected, Erb's traumatic form of shoulder paralysis should be excluded. Some cases closely resemble this form of paralysis. If the paralysis occurs immediately after birth and follows traction on the arms, poliomyelitis may be excluded. If the paralysis occurs after the sixth month, the diagnosis, in the absence of any traumatic history, should be that of poliomyelitis.

**Morbid Anatomy.**—The theory of Charcot, that anterior poliomyelitis is a primary degeneration of the ganglion-cells in the anterior horns of the gray matter in the cord, has given way to the belief that there is severe inflammation superinduced by some toxic agent circulating in the blood. The change begins in a degeneration of the bloodvessels of the anterior median fissure. There is proliferation of the endothelial lining of these vessels. The inflammatory process extends to the surrounding neuroglia and the ganglion-cells supplied by those vessels in the anterior horns of gray matter. In severe cases, the motor nuclei of the medulla may be involved. There may be inflammatory exudation and hemorrhage. In recent cases, the ganglion-cells show granular swelling, vacuole formation, hyaline changes, disintegration, and atrophy. After months, there is paucity of ganglion-cells in the region corresponding to the paralyzed members. They are seen in a few groups in the anterior horns or may be entirely wanting. Outside of the affected area, there may be a diminution of the number of ganglion-cells throughout the whole cord. The nerve-fibres corresponding to the ganglion-cells which have disappeared are also wanting. There may be no marked change in the glia tissue, in the transverse section of the anterior horns, and in the general configuration of the transverse section of the cord. After a time, however, there will be sclerosis and atrophy of the affected horn. The sclerosis may affect the white columns. The anterior horns and corresponding white substance may be transformed into a glia tissue resembling gelatin, the spaces containing fluid granules and disintegrated nerve-tissue

(Ziegler). All these changes point to permanent injury to the spinomuscular neuron, the ganglion-cell of the anterior horn, and its nerve-fibre.

The **treatment** of anterior poliomyelitis is symptomatic. In the stage of onset, perfect rest and quiet are indicated, and a few remedies to meet the symptoms. The bowels are kept open with calomel. Bromides are used if the patient is restless. Ice applied to the nape of the neck or to the head, as in cerebral disease, is useful if there are cerebral symptoms, such as headaches. Ergot has been given to act on the blood-supply of the cord, but is of doubtful value. After paralysis is established and atrophy has made its appearance, massage of the affected muscles, and electricity, especially of the galvanic form, are indicated. Later, in the chronic stage, much can be done for the sufferers by orthopedic appliances, such as braces and splints. If there is contraction of opposing muscles, tenotomy should be resorted to. In cases in which the joints have become the seat of luxation, arthrodesis has been practised by surgeons with good results in increasing the power of the affected limbs.

#### **THE JUVENILE FORM OF PROGRESSIVE MUSCULAR ATROPHY (ERB'S TYPE).**

This disease is characterized by a weakness and progressive wasting of certain muscles. It begins in childhood or early youth, and involves, as a rule, the shoulder-girdle, the upper arm and pelvic girdle, and the thigh and back. The muscles of the forearm and leg remain for a time intact. This atrophy may be associated with true hypertrophy or pseudohypertrophy of some muscle. The pectoralis, the trapezii, the latissimi dorsi, the serrati, the rhomboids, the upper arm muscles and supraspinators, are apt to be wasted. The deltoids, supraspinati, and infraspinati may be normal or hypertrophied for a time. There are no fibrillar contractions, no disturbances of sensation, and no reactions of degeneration and visceral disturbances.

#### **THE LANDOUZY OR DEJERINE TYPE OF THE FACIO-SCAPULO HUMERAL FORM OF MUSCULAR ATROPHY.**

This form in no way differs clinically or pathologically from the juvenile form of muscular atrophy. Authors include in this class all cases in which the atrophy begins in early life, as a rule, in the muscles of the face. The patients have a peculiar expression—so-called "*facies myopathique*." The lips are thickened ("*bouche*



de tapir" or tapir mouth). The shoulders later become atrophied. The supraspinati, infraspinati, and the flexors of the hands and fingers remain normal, as do the muscles of deglutition, mastication, respiration, and the laryngeal and ocular muscles. There are no fibrillary twitchings. The spinal forms of progressive muscular atrophy differ from primary dystrophy in that the onset of the latter affection is in the upper extremities. The disease is not hereditary, and fibrillary twitchings and electrical reactions of degeneration are absent.

Both these forms are probably clinical varieties of the pseudohypertrophic form of paralysis.

### PSEUDOHYPERTROPHIC MUSCULAR PARALYSIS.

This disease is characterized by a progressive change in the size of many of the muscles of the body and by a diminution of their power. It was described by Duchenne in 1861. Since then the most notable work on the subject has been done by Gowers, of England, and Sachs, of this country. The male sex is more frequently affected than the female. From two to eight members of the same family are often affected. Isolated cases are uncommon. The disease frequently affects the members of one sex in a family group. It is congenital but not hereditary. The antecedent cases, if there are such, can usually be traced on the mother's side of the family. The mother may be herself unaffected. Intemperance does not seem to exert any influence on the occurrence. Gowers notes that frequent marriage of parties closely related tends to predispose to the development of the disease in the children. In one-third of the cases the disease appears when the child begins to walk, and in children who are late in learning. It may manifest itself in the mid-period of childhood. In another third of the cases the children are in apparently good health until the fourth or sixth year. Three-fourths of the cases show symptoms of the disease before the tenth year. The disease may not manifest itself until after puberty, and may only be noticed during convalescence from some intercurrent acute disease.

The **symptoms** are impairment of power and change in the form of groups of muscles or of single muscles. The impairment of power is at first not very apparent. The muscles of the calves enlarge, and show a very characteristic and significant hypertrophy. Mothers are at first pleased with what appears to be muscular development of the children (Gowers). It is then noticed that although the muscles of the calves and glutei are large, the children are easily fatigued in mounting stairs. They fall easily and rise

with difficulty. This loss of power is at first interpreted as weakness, but when it is found to be progressive the children are brought to the physician. The gait becomes pronouncedly oscillating. The body is inclined so that the centre of gravity is brought successively over each foot. In trying to rise from the ground the patient places a hand on each knee in a very characteristic fashion. By grasping the thighs and throwing back the weight of the trunk, the patient helps himself into the erect posture. The weakness of the muscles finally becomes extreme. The patients can neither stand, walk, nor sit upright. They become bedridden. In the early stage, the muscles of the trunk may be normal, small, or atrophied, and those of the lower extremities much enlarged. Single muscles or groups of muscles of the arm and forearm may be enlarged (Plate XXII.). Finally, as the atrophy and weakness increase, there are contractures and distortions of the extremities and trunk. Equinus, lordosis, and lateral curvature are very marked. The knee may become fixed and distorted by contractures. The muscles most frequently affected in the beginning are those of the calves of the legs. These sometimes attain an enormous size. Those of the anterior part of the leg are not so much enlarged. The flexors of the knee commonly escape. The glutei and lumbar muscles are enlarged. The infraspinatus muscle is frequently enlarged, and stands out prominently; it is often mistaken for the lower edge of the scapula. The deltoid is often large; the serratus and the pectoralis are rarely affected. The triceps and biceps are frequently large, but often only in parts. The muscles of the forearm suffer only in a minority of cases. The intrinsic muscles of the hand are never affected. In that respect the disease is sharply distinguished from atrophies of spinal origin. The muscles of the neck are, with the exception of the clavicular portion of the sternomastoid, rarely affected. All the muscles affected are weakened, the smaller and atrophied muscles more so than the others. There is reason to believe that many muscles not visible are much affected.

**Electrical Reaction.**—This is altered when weakness sets in. The electrical contractility to galvanic and faradic stimulus finally disappears.

**Reflexes.**—The knee-jerk is at first normal. It later diminishes and finally disappears. It is never increased in a pure case. In one case in my hospital service there were increased reflex at the knee and foot-clonus. This case gave a history of a blow across the back. Sachs, with whom I saw the case, suspected a complicating myelitis of the cord.

Sensation is unaffected and the sphincters remain normal.

The **course** of the affection is prolonged and tedious. The disease is progressive. It may be ten or fourteen years before the patients succumb. They die of some intercurrent disease. If the



PLATE XXII.



Pseudohypertrophic Paralysis in a Boy Eight Years of Age. Hypertrophy of the infrascapulari well shown; also atrophy of the muscles of the thorax and hypertrophy of the glutei and the muscles of the lower extremity.





disease appears after puberty, the course is slower than in cases in which the first symptoms are noted in early childhood.

**Varieties.**—There are cases in which only one muscle or group of muscles of the extremities is enlarged, the others being small or normal in size. There are other cases in which all the muscles are small and waste progressively.

**Complications.**—Chorea, poliomyelitis, myelitis, mental deficiencies, and epilepsy may complicate the affection.

**Morbid Anatomy.**—The gray matter of the cord and the nerves are normal in appearance. There may be slight hemorrhages. The neuroglia-cells have sometimes been found to be increased. The disease is, however, primarily one of the muscle-tissue. The muscles are pale-yellow. They are replaced mainly by fat and connective tissue. The muscle-fibre is narrower than is normal, although in advanced cases the residual muscle-fibre may retain its transverse striation. Where the muscle-fibre is narrow it becomes granular or is the seat of fatty or waxy degeneration and vacuolization. Empty sarcolemma-sheaths are seen.

The **diagnosis** is made from the progressive weakness, the gait, and the mode of rising from the recumbent position. The peculiar enlargement of the muscles of the calf and infraspinatus, the atrophy of the latissimus dorsi and lower part of the pectoralis, and the immunity of the intrinsic muscles of the hand are characteristic. In the stage of contracture, this disease differs from congenital spastic paraplegia in that there is no increase of deep reflexes.

The **prognosis** in children is grave. The affection is progressive.

**Treatment.**—Much can be done for the patients by means of massage and electricity. In the stage of contractures, while there is still power, relief can be secured by tenotomy.

## DEFORMITIES OF THE SKULL AND SPINAL CANAL.

These deformities do not strictly belong to the diseases of infancy and childhood. Only the forms most commonly met are here considered.

The faulty closure of the spinal canal causes a deformity called rachischisis or spina bifida. If the defect involves the spinal canal in its whole extent, there is rachischisis totalis. The vertebrae form a shallow canal in which lies the rudimentary spinal cord covered with a thin membrane. If the defect of the bony canal is only partial, there being a sac-like protrusion of the cord and its membrane, there is said to be a rachischisis cystica or spina bifida cystica or rachicele.

Faulty development of the cranial bones with rudimentary brain

is called *cranioschisis* (Fig. 147). If with the cranial defects there are defects of the bony vertebral canal, there is said to be *cranio-rachischisis*.

If there are only partial defects in the cranial bones, with sacculated protrusion of the membranes of the brain (pia and arachnoid), with fluid in the sac, there is a *meningocele*. *Meningo-encephalocele*

FIG. 147.



*Cranioschisis.* Deficiency of the frontal, parietal, and most of the occipital bones. Protrusion of the cranial contents in shape of a sac covered by hair and scalp, and containing fluid and brain substance. Blindness; idiocy.

is a sac containing in addition the brain-substance. *Encephalocele* is a hernia of the brain and pia, no fluid being present in the sac.

### **Spina Bifida.**

*Spina bifida* or *hydrorrhachis* is a congenital deficiency in the vertebral laminae, through which the cord and its membranes protrude in the form of a sac containing fluid. The deformity is most frequently seen in the dorsolumbar, dorsosacral, and cervical portions of the vertebral canal. It rarely occurs in the middorsal region. It is generally single. It may occur both in the neck and in the lumbar region.

The tumor may be small and only indicated by a fissure, or may, as in Broca's case, attain a circumference of 62 cm. It may be flat or pedunculated. The latter form is uncommon. The surface of the tumor may be smooth or lobulated and uneven. The lobulated forms indicate divisions in the interior of the sac. The skin covering



the sac may be very thin or glistening. It may burst during delivery, may be thick and vascular, or covered with cicatrices and granulating ulcers. In some tumors the subcutaneous tissue can be made out; in others the skin is atrophic. In rare cases the tumor is composed of a mass of mucous tissue situated between the skin and dura mater. In the interior of this mass there is a small cavity (Kirmisson). Von Recklinghausen and Muscatello have demonstrated that the statement that the sac of the spina bifida is lined with dura mater is incorrect. Hildebrandt has, however, found cases in which the dura lined the sac. The pia and arachnoid line the sac. The fluid in the sac is serous and colorless or lemon-colored. It is alkaline in reaction, rich in salts, and contains sugar. If inflammation is present, blood is found in the sac. The fluid is either outside the cord or in the central canal (Virchow).

Spina bifida is, with reference to the nature of the contents of the sac, divided into three forms:

(a) *Myelomeningocele*, in which the fluid in the sac is situated between the cord and its membranes.

(b) *Meningocele spinalis*, in which the inner surface of the sac is formed by the arachnoid and pia mater.

(c) *Myelocystocele*, in which the fluid is situated in the central canal of the cord.

The *myelomeningocele* forms a broad but not very prominent tumor, which may be found in the lumbosacral, cervical, thoracic, or sacral regions. At its base the tumor is reddish, and is covered with fine, long hairs. This zone is from 1 to  $1\frac{1}{2}$  cm. broad. In the centre of the tumor there is a reddish-brown velvety vascular area, the remains of the medullary vascular zone. The sac is formed of arachnoid and pia mater. Its interior is crossed by nerve-trunks. The cord is drawn outward and some nerves may arise from the prolongations of the cord. Accordingly, there is an accumulation of fluid in the meninges (*hydromeningocele*), with an accompanying hernia of the cord (*myelocele*).

*Meningocele spinalis* is the rarest form of spina bifida. The sac is composed of pia and arachnoid. The latter may be much thickened. The opening into the vertebral canal if large may allow hernia of the cord. If the tumor is situated in the sacral region, the interior of the sac may contain the nerves of cauda equina.

*Myelocystocele*, *hydromyelocele*, or *syringomyelocele*, is that form of spina bifida in which there is a dilatation of the central canal of the cord. The dura is lacking in the sac, which is lined with cylindrical epithelium. The spinal cord in part of its extent may be found in the sac, or may be found on the exterior wall of the sac and end there. It may break up into several bundles. In the interior the spinal nerves form a series of loops with their convexities posteriorly. They may return into the vertebral canal or may

end in the sac. Spina bifida is a primary agenesis. The growth of the sac is due to inflammatory processes.

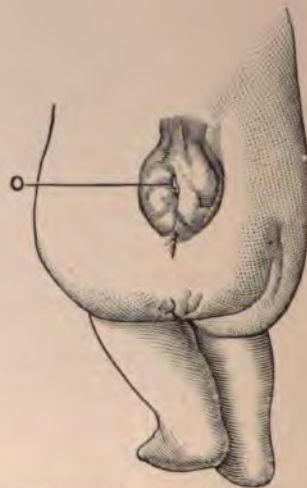
**Symptoms.**—The tumor is the chief physical sign. It is situated in the median line or may be at one side. It is round or elliptical and covered with thinned or thickened skin (Figs. 148 and 149). In the centre of the myelocystocele is a depression which gives the tumor a tomato-like appearance. The tumor may be soft, hard, or fluctuating. The defective vertebral laminae may be discerned on palpation. The tumor enlarges and becomes tense when the patient assumes the upright posture, cries, or exerts himself. When the

FIG. 148.



Spherical form of spina bifida lumbalis.

FIG. 149.



Elliptical form of spina bifida, with fistulous opening (o) into the vertebral canal.

patient takes the recumbent posture it becomes smaller. It also does so at each inspiration.

In some cases the functions of the individual are normal. In others, the mobility and sensibility of the lower extremities are affected. Deformities of the foot similar to those seen in infantile paralysis are sometimes present. There may be incontinence of urine and feces. There are sometimes trophic disturbances, such as perforating ulcers. These are of value in the diagnosis of lumbar tumors which are apparently lipomatous in their nature and are covered with hair (Kirmisson). In such tumors, disturbances of sensibility occurring with perforating ulcers and deformity and atrophy of a lower extremity are significant of spina bifida.

**Course.**—Spina bifida if left to itself may grow to a large size, may burst or ulcerate, and cause death by pyogenic infection of the



meninges and cord tissue. In other cases a lineal ulcer discharges fluid and closes up several times in succession. In some cases of spina bifida the tumors remain stationary in size until late in adult life. In rare cases spontaneous cure results by inflammation of the pedicle of a pedunculated spina bifida.

The **diagnosis** of spina bifida is not difficult if what has been detailed of the anatomy and symptomatology is borne in mind. Muscatello gives the following characteristics of the various forms:

FIG. 150.



Spina bifida lumbalis, with pes valgus on the right side; also congenital subluxation of the hip.

FIG. 151.



Spina bifida occulta pes calcaneovalgus on the right side; pes equinovarus on the left side.<sup>1</sup>

In **myelocystocele** there is a round tumor with a wide base. The tumor is lumbosacral, elastic, translucent, and fluctuating, and does not diminish on pressure. Pressure causes tenseness of the fontanelle. There may be scoliosis, lordosis, abdominovesical fissure, and deformity of the foot.

In **myelomeningocele** there is a flat, soft, elastic tumor, either lumbar, sacral, cervical, or thoracic. It may be complicated by

<sup>1</sup> Figs. 148-151 are from Kirrison.

umbilical hernia, paralysis of the extremities and bladder, and deformity of the foot.

In **meningocele** there is a sacral pedunculated translucent tumor, but no disturbances of mobility or sensibility.

Of considerable interest is the form called **spina bifida occulta** (Figs. 150 and 151). In these cases there may be no tumor, the seat of the deformity being indicated by a depression or dimple. In other cases, as in that shown in the illustration from Kirmisson, there is a small tumor of doughy consistency on one of the gluteal folds. The tumor may present an umbilication. Spina bifida occulta should be suspected in cases in which abnormal sacral depressions or tumors occur in connection with clubfoot deformities or congenital incontinence of urine or feces, or of both.

The **treatment** of spina bifida belongs to the domain of surgery. The treatment by injections of Morton's fluid (2 per cent. of iodine, 6 per cent. of potassium iodide in glycerin) has been abandoned in favor of excision of the sac.

#### LEADING AUTHORITIES REFERRED TO IN CHAPTER VIII.

*Boltze, Otto*: Zur operativen Behandl. des Hydroceph. Chr., Thesis, Halle, 1893.  
*Crozer Griffith, J. P.*: "Tetany in America," American Journal Medical Sciences, 1895.

*Griffith*: Hereditary Ataxia, Amer. Jour. Medical Sc., 1889.

*Epslein, A.*: "Kataleptische Erscheinungen," Prag. med. Wochen., 1896.

*Escherich, T.*: "Tetanie im Kindesalter," Berlin. klin. Wochen., 1897.

*Fischl, R.*: "Tetanie, Laryngospasmus u. Rachitis," Verhandl. d. Ges. f. Kinder., xiii.

*Hochsinger, C.*: Myotonie der Sanglinge Wien, 1900.

*Hirsch, Wm.*: "Pathological Anatomy of Amaurotic Family Idiocy," Journal Nervous and Mental Disease, 1898.

*Ireland, Wm. W.*: The Mental Affections of Children, Philadelphia, 1900.

*Kirmisson, E.*: Lehrbuch Chirurg. Erkrank., Stuttgart, 1899.

*Macewen, Wm.*: Pyogenic Infectious Diseases of Brain and Spinal Cord, 1893.

*McNutt, S.*: "Apoplexia Neonatorum," Amer. Jour. Obstet., etc., 1885.

*Olser, Wm.*: Chorea, Philadelphia, 1894.

*Pfaundler, M.*: "Lumbalpunktion," Beitrag zur klin. Med. u. Chir., Heft 20, 1899.

*Rotch, T. M.*: "Rhachischisis," Trans. Amer. Ped. Soc., 1900.

*Rüttimeyer*: Virch. Arch., Bd. 90.

*Sachs, B.*: Nervous Diseases of Children, New York, 1895.

*Sachs, B.*: "Amaurotic Idiocy," Deutsch. med. Woch., 1898.

*Säenger, A.*: Neurasthenie u. Hysterie bei Kindern, Berlin, 1902.

*Thomson, J.*: "Infantile Respiratory Spasm," Edinburgh Medical Journal, 1892.

— "Congenital Stridor," British Medical Journal, 1906.



## CHAPTER IX.

### GENERAL DISEASES.

#### **RACHITIS.**

(*Rickets.*)

**RACHITIS** is a disease of nutrition causing well-marked changes in the structure and form of the growing bones. It is peculiar to infancy and childhood, and does not occur after the skeleton is formed.

**Etiology.**—There are two forms of rachitis, the congenital or foetal and the post-natal.

The occurrence of congenital, foetal, or intra-uterine rachitis is still a subject of much difference of opinion. According to some authorities (Kassowitz), 80 per cent. of the infants of the Vienna Maternity Hospital show evidences of rachitis. Epstein at one time demonstrated the great frequency of rachitic deformity at the costochondral junction of the ribs, in the infants of the Maternity Hospital in Prague. On the other hand, Monti, Guerin, and Virchow insist that foetal rachitis in the true sense is rare, and that an anomaly in the development of the primordial cartilage has been mistaken for rachitis, with which it has nothing in common.

There are other forms of disease, such as achondroplasia, which have been described as foetal rachitis by Thompson, of Edinburgh, and Townsend, of Boston. These cases seem to be allied to cretinism and are classified by some authors as forms of it, but have nothing in common with that condition.

Hemorrhagic rachitis is a term applied by some authors to Barlow's disease or infantile scurvy. Rachitis is for the most part post-natal, and its onset occurs most frequently during the first year of life. It is rare after the third year. The sexes are equally subject to the disease. A moist climate favors it. It is very common in Germany and Austria, and is rarely met in Southern Asia or Central America. Fischl insists that it is peculiar to some races of people, and Snow, of Buffalo, has shown that Italians living in America, are peculiarly subject to it. It is most common among civilized communities, in which infants, especially those of large cities, are fed upon substitutes for breast milk. On the other hand, breast-fed infants may develop rachitis, but in such cases investigation of the milk by Pfeiffer and others has not resulted in the dis-

covery of any peculiarity of the milk which might be looked upon as a causative factor. Rachitis develops in infants who have been weaned from the breast early and fed on artificial foods or sterilized milk. The early introduction of meats and solid food into the dietary of the infant has been cited as an etiological factor.

That syphilis is a direct causative agent in rachitis (Parrot) can no longer be accepted. Heredity does not seem to exert any influence. There are many theories as to the active and immediate causes. The principal theories are those which presuppose the lack of some element, such as phosphates or lime salts, in the food, and those that trace the processes of rachitis to a disturbance of nutritive functions caused by an increase of certain acids (lactic) in the stomach, a diminution of others (hydrochloric) and resulting intestinal functional irregularities (Monti, Zander). The intestinal disturbances cause the elimination of certain salts from food, hence the blood fails to receive what is necessary for the structure and formation of the bones.

**Morbid Anatomy.**—Rachitis is anatomically characterized by processes which cause an increased resorption of bone, deficient calcification of cartilage, and the formation of a characteristic tissue—a deficiently calcified bone, the so-called osteoid tissue (Ziegler, Kassowitz, Schmorl). The increased resorption consists in an augmentation of the number of areas of lacunar absorption. In marked rachitis the greater part of the bony skeleton is lost. The cortical area of the long and of the short bones becomes osteoporous. A large part of the lamellæ of the cancellous bone is absorbed and disappears. In the flat bones the arrangement of outer and inner table separated by the intervening diploë is lost. The bone tissue is reduced to a few lamellæ. At the zones of periosteal and medullary ossification, the lamellæ are replaced by osteoid tissue. This tissue is a new formation devoid of lime salts.

The marrow of the osteoid tissue formed from the periosteum or medullary canal consists of a reticulum of striated connective tissue rich in bloodvessels and enclosing free round cells. Beneath the periosteum of the cranial and long bones there is formed, because of these changes, a spongy vascular tissue which is resistant to pressure and may be cut with a knife. While the rachitic process lasts, no lime salts appear in the lamellæ of osteoid tissue, but as soon as the disease has spent itself those salts appear in the centre of the lamellæ. Complete recovery results in calcification of these lamellæ, which being proliferated leave the bone hardened and very much thickened. The pathological change in the endochondral ossification consists in an entire absence of a calcification zone. In severe rachitis, all signs of the deposit of lime salts are absent. There is a widening of the zone of proliferation of cartilage cells, and also of the columns of hypertrophoid cartilage cells. There is lastly an irregular forma-



tion of vascular marrow-spaces, which grow here and there into the cartilage from the bone. Thus at the junction of cartilage and bone, there is in the long bones no distinct line of ossification. The red marrow-spaces extend for varying distances into the cartilage.

The abundant growth of bloodvessels extending from the perichondrium into the cartilage is accompanied by the substitution of osteoid tissue and marrow-spaces for the cartilage proper, as in periosteal and medullary ossifications. In rachitis the cartilage is never completely absorbed by osteoid tissue. Thus, on section, the bone shows, nearest the cartilage, the zone of proliferating cartilage-cells with hypertrophied cells in columns; next to this is the zone of osteoid tissue in lamellæ in which few lime salts are deposited. Nearer the bone are lamellæ of osteoid tissue, in the centre of which fully formed bone is deposited. The lamellæ of osteoid tissue differ from those of normal bone in being much thicker and more abundant. The osteoid tissue is very resilient and easily bent, hence this property of rachitic bones. The process leaves the bones much thickened, especially at the epiphyseal extremities. The deformities of the chest, extremities, pelvis, and spine can thus be traced to the tendency of the rachitic bone to bend on pressure and traction. The effects of the process on the shape of the cranium and the delay in the formation of the teeth may thus be easily accounted for.

Among other gross lesions connected with the clinical picture of rachitis is enlargement of the spleen. The organ may be very large and easily palpated below the border of the ribs. In such cases the liver may also be apparently enlarged. During life the enlargement of the liver may be more apparent than real. The chest, if narrow and deformed, may cause downward displacement and rotation of that organ. In rachitic infants the lymph-nodes are more apparent on palpation than is normal. They, however, are never increased to the size attained in tuberculosis, syphilis, or eruptions of the skin, such as those of the exanthemata. The blood may show the changes of extreme simple anæmia—an increase in the nucleated red blood-cells and other signs.

**Brain.**—Slight or marked hydrocephalus is frequently found in rachitis. The relation between the two conditions is not clear. If the infant dies of an intercurrent disease, changes of a chronic catarrhal character may be found in the gut and signs of bronchitis or persistent bronchopneumonia in the lungs. These conditions follow the changes in nutrition which cause the rachitic processes elsewhere.

**Symptoms.**—The most marked and general symptoms of rachitis are changes in the bony skeleton.

**The Head.**—The shape of the rachitic head is very characteristic. The frontal bone bulges, giving the infant a very prominent forehead. The parietal bones have a flare, caused by the formation of bosses at the centres of ossification. The whole head has a cuboidal

shape, which, with the proportionately small face, gives the characteristic appearance. The disturbances in bone formation cause the appearance of soft spots, especially in the vicinity of the lambdoidal suture. These (craniotabes) may be membranous in structure. They rarely appear on the frontal bones in the vicinity of the coronary suture. The spots of craniotabes appear in infants who develop rachitis before the sixth month (Monti), rarely after this period. They take four or five weeks to develop fully. In developed rachitis the occiput is flat and devoid of hair (Plate XXIII.). The anterior fontanelle,<sup>1</sup> which normally closes between the fifteenth and the eighteenth month, remains open for a long time, in some cases until the third or fourth year, or even to the sixth. The sutures are also slow in closing. The coronary sutures may remain open for two, and the longitudinal suture for three years. The lambdoidal suture does not in some cases close until the eighteenth month.

If the thorax is affected by rachitis, the circumference of the head will exceed that of the chest. The lower jaw has an angular deformity, described by Fleischmann. This consists in a bending of the body of the jaw at the situation of the canine teeth. The body of the jaw is also rotated internally on its horizontal axis. If rachitis begins before the sixth month, dentition is delayed for periods varying up to a year and a half. I have a record of a case in which the first tooth appeared at the twenty-fourth month. If rachitis develops after appearance of the first teeth, the succeeding ones appear later than is normal. The structure of the teeth suffers. They show erosions, are easily broken, and become carious quickly. This is due to imperfect formation of enamel or dentine. Some time after their eruption, the incisors show a well-marked incurvation at the free border, which is due to erosion or breaking of the tooth.

The **thorax** shows very characteristic deformities. Rachitis of the thorax in most cases develops in the second half year, and may continue into the third year. The first marked sign is the appearance of the so-called rib rosary. This is a thickening of the costochondral junction of the rib, in which the rachitic processes above described are very active. Deformity of the thorax follows in course of time. The thorax becomes prominent at the sternum and flattened in the midaxillary region from the axilla to the free border of the ribs. There is a distinct incurvation of the thorax above, and a flaring below. The thorax is much narrowed at the clavicles, with a flaring outward of the lower ribs. Respiration, especially inspiration, is much interfered with. The sides of the thorax are drawn

<sup>1</sup> While the lateral and posterior fontanelles close during the first months of infancy, the anterior fontanelle increases in its longitudinal and transverse diameter with the growth of the cranium up to the twelfth month. The growth of the anterior fontanelle was first observed by Elsässer. Although denied by Kasowitz it has been recently proved by Rhode that the contention of Elsässer is correct.



PLATE XXIII.



Rachitis. Showing the cuboidal shape of the head, the thoracic deformity, the beaded ribs, the protuberant abdomen, and the enlarged lower end of the radius.





inward at the diaphragm at each inspiration. In an attack of severe bronchitis or bronchopneumonia, the drawing inward of the sides of the chest becomes still more marked. In some cases the sternum alone is affected. There is a sinking of the sternum, with resulting chest deformity. Some forms of rachitis affect only the ribs or part of the thorax. While the rachitic process is in progress, the chest circumference does not increase; it begins to do so when the disease has run its course in the thorax.

FIG. 152.



Rachitic deformity of the spine Uniform curvature backward.

**Pain.**—When the infant is raised from the chair or crib, it cries. This is the result of the painful nature of the rachitic process in the bones. Forceful percussion of the chest will cause pain. On account of the deformity of the chest and the consequent interference with its physiological functions, the lung is prone to contract infections, such as bronchitis and bronchopneumonia. Atelectasis is also a common complication. The clavicle becomes bent and fractures on the slightest traumatism. At the termination of the rachitic

process, the clavicle and scapulæ are much thickened. Virchow has shown that the scapula becomes the seat of an angular deformity.

**Spine.**—On account of the relaxation of the ligaments of the bodies of the vertebræ and of the rachitic processes in the bodies of the bones themselves, there is in most rachitic infants a bending backward of the dorsolumbar spine (Fig. 152). The curvature is very marked

FIG. 153.



Angular deformity of the spine, due to Pott's disease, as distinguished from the deformity due to rachitis.

when the infants are held in the arms. It differs from deformity due to Pott's disease in that it is not angular, and in that the



spine can be straightened and even curved forward with ease (Fig. 153).

Lateral curvatures of the spine are also found. If the spinal deformities occur early in infancy, they disappear as the rachitis heals and the ligaments and muscles regain a normal tonicity. On the other hand, should the rachitic process attack the spine late in the third or fourth year, the deformities are perpetuated. This is especially the case if the pelvis is also affected at that time (Monti).

The **pelvic deformities** which result from rachitis are chiefly flattening of the pelvis, and the pseudo-osteomalachic pelvis.

**Upper Extremities.**—The epiphyses are much swollen and, in rare cases, painful. The wrist is flat and much broadened. If the

FIG. 154.



Marked general rachitis in a child thirteen months of age. The phalanges of both hands thickened, simulating dactylitis syphilitica. Other osseous deformities present characteristic of rachitis. No syphilis.

rachitis is elsewhere not marked, the physician should be careful not to mistake a normal enlargement in this situation for rachitis. In exceptional cases, the elbow and shoulder-joint show similar changes.

On account of the traction of the flexors and pronators, the forearm may be incurvated and the bones twisted on their longitudinal axes. The result is a more or less fixed position of pronation in the forearm. The arm is rarely curved in this manner, but it may, like

the clavicle, be fractured after slight traumatism. As a result of rachitis and deformity, the growth of the bone in length is much interfered with.

The phalanges are sometimes the seat of the rachitic processes. In one severe case I found all the phalanges thickened in the diaphyses. This case bore a very close resemblance to dactylitis syphilitica, especially as there was pain on pressure (Fig. 154).

The deformities of the **lower** extremities are more marked than those of the upper ones. On account of the pain experienced, the infants refuse to stand; they will draw the extremities up underneath the abdomen, if any effort is made to make them do so. In other cases, when attempts are made to stand, the weight of the body and the muscular traction (Kassowitz) cause deformity. The femur, tibiae, and fibulae curve outward, giving the so-called "bow-leg" deformity (Plate XXIV.). This may in extreme cases result in a deformity of the heads of the bones entering into the formation of the knee-joint. The ankle-joint may suffer a varus deformity. The femur and tibiae may curve inward, and a knock-knee deformity result. In all cases, there is relaxation of the ligamentous joint-structure. The tibia sometimes becomes much thickened and curves anteriorly, giving the so-called "sabre deformity." It may be twisted on its longitudinal axis. I have seen severe rachitis of the femur and tibia result in multiple fractures.

The deformity at the hip-joint, which later in life follows changes in the angle made by the neck of the bone with the shaft of the femur (coxa vara), is believed to be due (Whitman) to rachitis. The children are late in walking. The musculature is weakened through disuse.

When the children assume the sitting posture, they cross the lower extremities in tailor fashion. In the majority of cases of rachitis, the abdomen is protuberant. As a result of the defective nutrition, the musculature of the gut is weakened in the same manner as that of the extremities. Tympanitic distention is the rule.

**Intestinal disturbances** are common in rachitis, but are not a result of the process. Hensch shows that rachitis may be present with an apparently normally functioning intestine.

The **spleen** is enlarged in many cases of rachitis, but retrogrades to the normal size after the disease has run its course.

The **blood** shows the changes found in ordinary mild or severe simple anæmia.

The **liver** may be slightly enlarged.

**Anæmia** of the skin and mucous membranes is frequently found. It may be so extreme as to cause the skin to have a yellowish waxy hue. Rachitic children perspire freely at night, especially about the head. Unless the skin is kept scrupulously clean, sudamina, furuncles, and eczema of all kinds will result.



PLATE XXIV.



Rachitis. Showing the deformity of the thorax, and marked bowing of the tibiæ.





**Nervous System.**—There is no doubt that certain nervous affections, such as tetany, laryngismus stridulus, attacks of inspiratory apnoea, spasmus nutans, and the so-called barn-yard crowing or congenital stridor of the larynx (described by Thomson), occur most frequently in subjects of rachitis. Some authors (Kassowitz, Jacobi, Escherich) trace a distinct etiological connection between these conditions of instability of the nervous system and rachitis.

**Hydrocephalus** occurs in rachitic subjects. In cases of severe rachitis, an appearance of mild hydrocephalus is given to the face by a downward depression of the eyeball. The sclera of the eyes is thus slightly exposed. The appearance seems to be caused by a depression of the orbital plates of the frontal bone by the overlying frontal lobes of the cerebrum. In many cases of severe rachitis, the wide fontanelle, its tenseness, and the open coronal and temporal sutures give a picture like that of a non-progressive, mild hydrocephalus which is simply a feature of the nutritive disturbances taking place in the brain as elsewhere.

**Severity of the Affection.**—These symptoms are not present in all cases of rachitis. In some cases there are only very slight signs of the disease, such as a slightly cuboidal shape of the head or a scarcely appreciable bending of the ribs without any deformity. In such cases even an expert may be in doubt as to the presence of swelling of the epiphyses. In other cases an intercurrent affection, such as tetany, will cause the physician to seek for signs of rachitis, which may be so slight as to have previously escaped notice. Craniotabes is sometimes absent in marked cases. Delayed dentition is not the rule. Rachitis may be very evident in cases in which the teeth appear in their normal order.

**Duration.**—In such a disease as rachitis it is to be expected that the duration of the affection will vary greatly in different subjects; it may last months in some cases, in others years. The first favorable sign is the attempt of the infant or child to walk, but children with marked and progressive rachitis sometimes walk early.

Increase in weight and in the chest circumference, an improvement in symptoms, such as anæmia and intestinal disturbances, and the cessation of pulmonary complications are indications that the disease has come to a standstill.

The **diagnosis** of rachitis before the development of the physical signs in the bones of the head, chest, and extremities is scarcely possible. Monti thinks that an increase of lactic acid in the stomach contents is, if there are intestinal disturbances, strong presumptive evidence of early rachitis, but the increase of lactic acid may be temporary, and the general practitioner will find it hard to estimate. Once the bone symptoms develop, there is no difficulty. In cretinism, Mongolian idiocy, and syphilis, there are changes in the bones which very closely resemble those seen in simple rachitis. Yet in all these

conditions there are other signs which will make the diagnosis clear. In syphilis, rachitis is an accompanying condition. There is no etiological connection between the two affections. In every case of tetany, spasmus nutans, laryngismus, congenital stridor of the larynx, inspiratory apnoea, or eclampsia, the physician should not fail to look for evidences of rachitis. The improvement in these conditions will often depend on the management of the rachitis.

If the infant cannot stand, the limbs may exhibit a variety of pseudoparalysis. Paralysis may be excluded by making an electrical muscle test. Although infants with rachitis will not stand, they move the lower extremities vigorously when lying down. This is not the case in the palsies; the faradic and galvanic muscle tests and the presence of the normal reflexes will fix the diagnosis. In severe cases of cranial rachitis, it is not always an easy task to exclude hydrocephalus. While marked hydrocephalus presents no difficulties, a slight hydrocephalus is not always apparent. In such cases the head circumference is measured once a month. An abnormal increase in the circumference, a wide tense fontanelle, and open sutures indicate hydrocephalus.

**Occurrence.**—West has demonstrated that rachitis in the United States is not confined to negroes and immigrants. He has shown that its greatest frequency is among the natives of Eastern Ohio.

**The Blood.**—Through a study of the blood in rachitis Morse has come to the conclusion that anaemia of any form may exist. It is generally an anaemia in which the number of red blood-cells is normal or nearly so. The hæmoglobin is reduced, and there is a consequent reduction in specific gravity. There is leucocytosis, especially in the cases with splenic enlargement.

**Rachitis tarda** is a term applied by Kassowitz and Genser to those cases which, instead of running their course in two or at most three years, continue in the active stage for eight, ten, or even twelve years. Kassowitz and his pupils record cases of florid rachitis at the tenth and twelfth year. I have seen a case of florid rachitis in a female child eight years of age. She had all the signs of rachitis of the head, thorax, and arms. The lower extremities were permanently crossed in tailor fashion. The bones were painful, and those of the lower extremities were the seat of multiple fractures. The teeth were decayed. In Genser's case the milk teeth having decayed and fallen out, the permanent ones failed to appear.

**Prognosis.**—If rachitis is not complicated by any intercurrent affection, the prognosis, even in the severe forms, is generally good so far as life is concerned. On the other hand, an intercurrent affection, such as pertussis or bronchopneumonia, is likely to run a severe course and prove fatal in a rachitic subject. If the rachitic process is complicated by nervous disorders, it is frequently fatal. Sudden death in eclampsia, tetany, or laryngismus is not uncommon.



The prognosis as to deformity will depend on the severity of the affection. Subsequent treatment will not always correct deformity of the pelvis and long bones. The conditions often remain permanent. Fortunately rachitis in this country is not among the native born of so severe a type as in Germany, Austria, and Switzerland. If marked hydrocephalus is a complicating condition, the prognosis is bad.

The **treatment** of rachitis differs greatly in different countries, but there are certain fixed principles upon which all methods are based. Prophylaxis is an important element in all methods. An infant at the breast should not be weaned too soon if the breast milk is sufficient in quantity and the infant is increasing in weight. Weaning should not be attempted until the ninth month. If it is done in the fall or winter, the milk should be obtained as soon as possible after the time of milking. There is no need of sterilizing the milk if it has been collected with care. It is at most pasteurized. Cows' milk should be diluted so that the albuminoid elements may be reduced. Articles of diet rich in albumins, such as eggs, should not be given early, nor should the infant be permitted to eat meat in any form, potatoes or vegetables. The early use of these articles of diet favors the development of rachitis. When the breast milk is insufficient, it should be supplemented by the requisite number of artificial feedings. Rachitic infants do better on two breast-feedings a day with several artificial feedings, than on artificial feeding alone. Cows' milk is the substitute for the breast. It should be properly prepared. Many severe forms of rachitis can be traced to the use of infant foods.

Artificially fed infants should, after the sixth month, be allowed a limited amount of fresh fruit juice once a day. Orange juice is best, but cannot be borne by all infants. An infant should not be allowed to become inordinately constipated. In other words, treatment is directed toward eliminating all predisposing factors to the development of the disease. Some breast-fed infants do not thrive. They develop serious disturbances of nutrition and colic, remain stationary in weight, and have irregular and green curdy movements. In such cases, the infant should be weaned or given another wet-nurse. Damp, ill-ventilated dwellings predispose to the development of rachitis.

**Bathing.**—Young infants should not be bathed in water which is much below the temperature of the body. Such bathing prevents increase in weight and causes disturbances of nutrition. The temperature of the bath should be practically the same throughout infancy. An infant cannot be hardened without disturbing the metabolism. The addition of sea salt to the bath water is advised by some physicians, and brine baths are in general use. There

are other kinds of baths which contain iron, but I have had no experience with them. They are not used in America.

Living at the sea-coast is believed to exert a very favorable influence upon rachitic infants and children. On the other hand, if there are affections of the chest and lungs, such as bronchitis of a chronic variety, the humid atmosphere of the coast is not likely to be beneficial, and mountain resorts are better.

**Medicinal Treatment.**—Cod-liver oil has long been a favorite drug in the treatment of rachitis. It should be given in the emulsion with the hypophosphites of lime and soda. An infant a year old should take half a teaspoonful three times daily. In intestinal disturbances, it should not be administered, for fear of aggravating the symptoms. The external application of the pure oil to the body can hardly be useful, since it certainly interferes with the metabolism of the skin.

IRON in the form of the hypophosphate, grain  $j$  (0.06) given four times a day, or the saccharated carbonate, grain  $ij$  (0.12) three times daily, is of great utility. The pomate of iron or the more digestible peptonates of iron and manganese are much used. The combination of thyroid extract and iron has, in some cases of extreme anæmia with enlarged spleen, been of great utility. I have used this combination only in cases where there was extreme anæmia with rachitis :

Thyroid ext. . . . .	gr. $\frac{1}{4}$ (0.03).
Sacch. carb. iron . . . . .	gr. $ij$ (0.2).
Tabes pulv. t. i. d.	

Henoch has advocated the use of thyroids in the advanced cases of rachitis. His view is opposed by other authorities (Monti). I advise the cautious use of thyroids in combination with iron in selected ambulatory cases only. Hospital cases will not do well on this therapy.

The lactophosphate of lime is advised by some authorities, but is of little value.

**PHOSPHORUS.**—It has been shown by Kassowitz and Wegner, and confirmed by Virchow, that in the lower animals phosphorus administered in sufficient dosage causes an increased activity in the processes at the epiphyseal ossification zone. The bone becomes more compact, but there is neither an increase of its diameter nor deformity. Kassowitz has contended that the same results are obtained in the human subject. On this question, there is wide difference of opinion. Jacobi was among the first in this country to administer phosphorus as a remedy for rachitis. He especially advises its use in cases of craniotabes. I have found that some children do well on it, while in others it causes gastric and intestinal disturbances. I have used the emulsion of lipanin, so much recommended by Kas-



sowitz, as a vehicle for the phosphorus. Enough of the phosphorus is put into the oil to make a teaspoonful of the emulsion equal to  $\frac{1}{280}$  grain (0.00024). Thompson's solution of phosphorus may also be used. Preparations of phosphorus, even those made with oil, deteriorate. Kassowitz advises the formula to be made up with recently dissolved phosphorus.

There are those who, like Henoeh, Monti, and Heubner, regard the phosphorus treatment of rachitis with distrust. The treatment of rachitis with glandular extracts is still a matter of empiricism. The treatment of the convulsions of laryngismus will be discussed in the section on that condition.

**Surgical Treatment.**—It is not within the scope of this book to dilate on the surgical or orthopedic management of rachitic deformities. It is, however, proper to state that it is neither right nor necessary to place every infant with marked spinal curvature due to rachitis in a plaster jacket. A young infant with marked backward curvature of the spine will gradually lose this deformity as its muscles improve in tonicity, but if placed in a plaster jacket will probably develop a subacute bronchitis or pneumonia. The lung is insufficiently inflated as it is, and becomes much more so if the soft thoracic walls and abdomen are encased in a plaster cast. In such cases the sitting posture should be avoided. The infants are kept in the arms or sleep on an ordinary hair mattress and hair pillow. It is not possible to keep them in any particular posture. Massage of the spine is of questionable utility.

Operations for the correction of deformities of the long bones should not be carried out until the rachitic process has come to a standstill. Surgeons sometimes advise the correction of deformities in young infants by encasing the limbs in plaster while the bones are still soft.

## RHEUMATOID ARTHRITIS.

(*Arthritis Deformans.*)

This affection should be sharply differentiated from all forms of chronic or subacute articular inflammation. Charcot and Weil have described this form of arthritis in children. The cases are not common. After the publication of my case, two others were described in the American literature, one of the descriptions being given by Manges. Cases of arthritis deformans or rheumatoid arthritis in children are referred to by Osler (4 cases) and Henoeh (5 cases). The onset of the disease is either sudden after an exposure to cold and wet, or slow. In one form, after an onset of chills and fever, soreness and pain in several joints appear. The child is at first able to be about, but, as the joints become more and more affected, complete disability results. The pain in the joints be-

comes so marked as to interfere with sleep. After a few months the patient may be unable to walk. In some cases the enlargements and pain begin in the lower extremities and gradually involve other joints. In others the onset is slow. The joints of the upper and lower extremities gradually become painful, and after repeated attacks remain swollen and limited as to motion. The ends of the bones are enlarged and there is effusion in some joints. With the progressive involvement of the joints there is atrophy of the

FIG. 155.



Arthritis deformans in a child seven years old. Deformity of all the joints with fixation. Child forced to assume this attitude awake and in sleep.

muscles, as in the adult form of the disease. When the disease is fully developed the condition is pitiable. In my case almost every joint in the body, including those of the cervical vertebræ, was involved; the temporomaxillary articulation, the shoulder, the elbow, the small finger-joints, the hips, knees, ankles, and toes, were all affected. The patient slept in a semi-upright posture, and had to be carried from place to place. There was very limited and painful motion in all the affected joints (Fig. 155). In



some cases there have been exophthalmic goitre and tachycardia (Manges); in others there also has been enlargement of the lymph-nodes, liver, and spleen.

Brabazon found that of 100 cases of this affection, only 3 per cent. occurred between the ages of five and fifteen years. Two theories have been advanced to explain this joint-affection; one, that of Charcot and Weil, is the neurotic theory, which is plausible because of the bilateral nature of the affection, the atrophy of the muscles around the joints, the changes in the skin which becomes in time tense and shining, and the enlargement of the ends of the bones which enter into the formation of the joints. The infectious theory is supported by the fact that there is in many cases a diurnal fluctuation of temperature of a degree or a fraction of a degree above the normal. The lymph-nodes are enlarged; the liver and spleen are also enlarged in some cases. The heart is not usually involved.

The **prognosis** as to life is good.

**Treatment** by massage, warm baths, and patient manipulation of the joints under anaesthesia, may effect slight improvement. In my case improvement was noted after a year of constant treatment. Iodide of potassium is the only drug which relieves the pain. In some cases it exerts a favorable influence upon the course of the disease.

### ACUTE ARTICULAR RHEUMATISM.

(*Polyarthritis Rheumatica; Rheumatic Fever.*)

Although acute articular rheumatism is still regarded by some authors as a constitutional disease caused by disturbances of nutrition which result in local manifestations, the general tendency is to regard it as an acute infectious disease. The infectious agent, whether bacterial or toxic, attacks the serous cavities, such as those of the joints, the pericardium and endocardium, and the pleura. The resemblance of rheumatism, especially in children, to the infections is sufficiently great to warrant a serious consideration of this theory. Thus in septic endocarditis in children, as in the adult, there are symptoms of pain in the joints. Chronic cases of endocarditis of a rheumatic nature in course of relapse occasionally take a septic course. Certain diseases, such as erythema nodosum and peliosis rheumatica, in which the joint-symptoms are marked, are regarded as being caused by infection of a bacterial nature. I have lately seen such a case of peliosis. In other diseases, such as scarlet fever, measles, and varicella, there are joint-affections which are recognized to be of an infectious nature. Lastly, both American (Packard) and English writers have called attention to the well-observed clinical fact that there are forms of rheumatism and endocarditis which follow attacks of tonsillitis of the lacunar type

or accompany them. It is true that the infectious agent, whether bacterial or toxic (Chvostek), is still to be discovered. Time may show that not one, but a variety of micro-organisms are capable of causing rheumatism of the acute articular type in a susceptible organism. Streptococci have been found in the exudate of the joints (Hlava). Staphylococci aureus, citreus, and alba have been found in the blood (Gutmann, Tizzoni, Bouchard). The pneumococci of Fränkel and the Diplococcus tenuis have been found in the joints (Leyden). Singer has found similar micro-organisms in the urine.

Heredity is among the predisposing causes. Children whose parents are markedly rheumatic, may suffer severely from the affection. Cold and exposure certainly predispose to the disease or precipitate attacks. The disease is prevalent in countries, such as England and America, in which climatic influences are favorable to its development, and is especially prevalent in the moist and cold seasons of the year.

**Age.**—Rheumatism has been described as occurring in early infancy (Jacobi). I have published a case in an infant of nine months. Rauefus, Chapin, and others have also described cases in infants. These cases were collected by Miller, who, with his own case (nine months), found in the literature only 19 authentic cases in nursing infants. Although rare in infancy, rheumatism is not uncommon in children from the fifth to the tenth year. The majority of the cases of rheumatism occur between the tenth and the twentieth year.

**Sex.**—Among adults, males are more subject to the disease. In children, however, although certain observers contend that it is more prevalent among girls, other statistics show that it has the same frequency of occurrence in the sexes.

**Symptoms.**—Certain peculiarities, pointed out by Jacobi, seem to differentiate acute articular rheumatism of infants and children from the same affection in adults. Not many joints are attacked. The pain and swelling are generally not very marked. The redness of the joint is slight or altogether absent. The temperature is rarely high. The smaller joints, such as the maxilla, sternoclavicular articulation, and those of the vertebræ, are rarely attacked. The larger ones, such as the ankle-, knee-, and wrist-joints, are most commonly affected.

Cardiac complication is the rule. As Jacobi has pointed out, endocarditis is sometimes the first manifestation of the disease.

**Clinical Types.**—In infants and young children the first signs are swelling and pain in the affected joints. The infant in the nursing period cries, has fever, and is restless. On investigation it is found that the patient favors one extremity, and shrieks with pain when it is touched. Children of two and one-half years or more refuse to



walk, and will complain of the affected joint, ankle, or knee. There will be fever and constitutional symptoms. The ankle, and in some cases the smaller joints of the foot are swollen. One of the knees, the wrist, and elbow may also be swollen, red, and painful. The fever rarely rises above  $103^{\circ}$  or  $103.5^{\circ}$  F. ( $39.4^{\circ}$  C.). In other cases there are fever and restlessness, and sometimes pains of an indefinite character in the joints. A history of pain may be elicited by careful questioning and examination.

The physician may find an angina, slight or marked; the heart may show signs of endocarditis of an acute type. There are pains in the joints but no true rheumatic swellings. The pains more closely resemble those in uncomplicated angina tonsillaris. In older children, with the endocarditis, a history of joint-pains may be obtained. In other cases, the pains in various joints are the only symptoms. There is no swelling or redness, and no endocarditis. Some cases have no fever. The classical cases, however, closely resemble those of the affection as seen in the adult. There may be premonitory symptoms, but as a rule the patient is brought to the physician with the enlargement of the joints fully developed. After the joints have become enlarged they may return to the normal in a few days, but may again be the seat of pain and swelling. The swelling in the joints of children does not persist as long as in the adult subject, and as a rule children are less disabled. In many cases there are gastric pains. The children do not show any greater tendency to perspire than adults.

**Endocarditis** is usually a complication of rheumatism in children. Its absence is rare. Only 2 of 15 of my hospital cases during the past year were free from cardiac complication. The most common cardiac lesion is found at the mitral valve and is manifested by a single systolic murmur at the apex. Three of the cases showed the presence of a double mitral murmur. Endocarditis sometimes does not reveal its presence by any symptoms, and is only discovered on a careful examination. In many of the cases there is also a pericardial friction first heard at the apex or base of the heart. The pericardial friction is more common in children than is generally supposed. The pericarditis frequently remains in the dry friction stage, and does not advance to effusion. Pleuritis and bronchopneumonia are among the less common manifestations. The endocarditis sometimes occasions pain and distress. The presence of endocarditis as an acute affection in first attacks of rheumatism has been dilated upon in the section on Endocarditis.

**Chorea.**—The relationship of chorea and rheumatism has been discussed. I have seen a child of two and one-half years born of a rheumatic mother, develop first rheumatism and endocarditis, and, within a few days, marked chorea. On the other hand, in many cases of chorea, there is neither endocarditis nor a history of rheu-

matism in children or parents. The statistics of chorea in hospital service show a greater frequency (39 per cent.) of cardiac disease with or without a history of rheumatism than the ambulatory cases. This is explained by the fact that only the severer cases of chorea come to the hospital.

The **prognosis** of acute articular rheumatism in infancy is good as to life. On the other hand, it is a disease which is likely to recur and to be complicated by endocarditis. The latter fact should cause the physician to reserve any definite prognosis until the course of the disease has been carefully studied. The prognosis of rheumatic endocarditis can never be definitely made. All depends on the amount of damage done to the valves and the frequency of the recurring attacks.

The **treatment** of acute articular rheumatism in children is not essentially different from that followed in the adult. I use salicylic acid, bicarbonate of sodium, salicylate of sodium, and oil of wintergreen.

The bowels should be kept open with an alkaline cathartic. The Carlsbad salt or Rochelle salt given daily is best adapted for this purpose. The patient is put on a milk diet; fruit juices are allowed. The patient is kept in bed. The affected joints, if painful, are either immobilized or wrapped in cotton. Some prefer to paint the joints with a solution of oil of wintergreen, and then wrap them in cotton. Salicylate of sodium is given internally in doses of grains ij to v (0.12 to 0.3) according to the age. Young children are given a dose every three hours. Older children are given doses of grains viij to x (0.5 to 0.6). The effect is watched. Salol or salophen may be given. The salicylates sometimes not only act as irritants to the stomach, but also have no appreciable effect on the course of the disease. In such I have given bicarbonate of sodium in increasing doses until the urine becomes alkaline. Endocarditis is treated on the principles laid down in the section on that disease. While under treatment the patient is given alkaline waters. During convalescence the various preparations of iron are of great value. The preparations of lithium are useful in cases in which there are indefinite pains in the joints. The carbonate is given in doses of grain j (0.06) three times daily. It is given in capsule to older children after meals. The method of treating rheumatic subjects by the occasional administration of salol or salicylates for months has been suggested. The salicylates upset the stomach, so that the alkalis alone are available. The patient is given grains v (0.3) of sodium bicarbonate twice daily every other day. Vichy water is used regularly. In some cases the *tablets* of vichy taken once or twice daily are of great value.



**Other Forms of So-called Rheumatism.**

(*Rheumatoid Affections.*)

There are forms of joint-affection which it is not yet advisable to class with true articular rheumatism, but which are constantly and incorrectly called rheumatic.

The **gonorrhœal** form of rheumatoid affection is seen in infants and children who suffer from gonorrhœal vulvo-vaginitis or urethritis (Koplik, Hartley, Moncorvo). It may be monarticular or many joints may be affected. It is not, as a rule, combined with endocarditis. I know of no such case in the literature.

**Peliosis.**—Cases of so-called peliosis rheumatica closely resemble acute articular rheumatism. I have seen several in older children. In one, there were for weeks repeated painful swellings of the joints, with purpuric eruption about them. The gastric pains and critical sweats so often seen in rheumatism were present. These cases rarely present a temperature above  $100.5^{\circ}$  F. ( $38^{\circ}$  C.). They show no cardiac lesion.

**Tonsillitis with Joint-pains and Endocarditis.**—Under the proper heading I have referred to cases of tonsillitis with indefinite pains in the joints and complicated with endocarditis.

**Erythema Nodosum.**—I have seen many cases of erythema nodosum in children. In all, the typical painful swellings on the anterior aspect of the tibia were present. There were also joint-pains, but in only 5 cases could I establish the presence of an endocardial murmur. I am therefore not willing to accept without reserve the contention of French authors that endocarditis is frequent in these cases.

The so-called **subcutaneous rheumatic nodules** are seen in children less frequently in this country than in England. They occur in endocarditis, and were present in 20 per cent. of Coult's cases (Donkin). They may be present in the absence of fever or in the febrile stage of rheumatism. They may be minute or of the size of an almond. They appear in crops, and may alternately appear and disappear for weeks. The nodules occur about the joints, elbows, knees, patella, over the vertebræ and scapula, and are freely movable under the skin which is not discolored. I have seen them in a case of rheumatoid arthritis, and also in one of peliosis rheumatica.

**Muscular rheumatism** is rare in infancy and childhood (Jacobi). Henoch describes cases of contracture of the muscles of the neck and of the nape of the neck. Among such contractures are forms of torticollis which are said to have a rheumatic origin. I have met many cases of torticollis in which with the contracture there was swelling of the cervical lymph-nodes. In such cases I have found eczematous affections of the scalp. It is possible that there was an acute infectious neuritis or myositis. There may, however, be cases resting on a purely rheumatic basis. All forms of torticollis due to

hematoma of the sternomastoid muscles or to cervical bone disease, glandular disease, or neuritis should be excluded before a definite conclusion is reached. Henoeh also refers to contractures of the abductors of the thigh which are of rheumatic origin. I have never seen cases of the kind.

### DIABETES MELLITUS.

Diabetes mellitus is of very rare occurrence in infancy and childhood. Simon says that he has met it in nurslings, but Monti doubts whether it can occur under the age of one year. In all his experience he has never seen such a case. Leroux, quoted by Monti, collected 147 cases of diabetes in children. The majority occurred between the fifth and tenth years. Of 159 cases collected by Saundby, 129 occurred between these years. Cotton has, in a recent article, shown that in children the ratio of deaths from diabetes to the whole death-rate is 0.04 per cent. in Chicago, and 1.2 per cent. in New York City.

The **etiology** of diabetes in children is practically the same as in the adult subject. Frerichs, Blanchard, Parry, and Roberts have shown that heredity plays an important rôle. In a case coming under my observation a sister of the patient had died of diabetes and four members of the family on the mother's side. In an instance reported by Roberts, 8 children of the family had died of it. It appears that in certain families there is a tendency to contract diabetes. There is no ground for assuming that diabetes in children follows traumatism or the infectious diseases, such as scarlet fever, measles, diphtheria, etc., any more frequently than in the adult. In some statistics, the sexes are shown to be equally affected. In others the disease is given as more prevalent in one or the other. Lemonnis has seen diabetes complicate congenital syphilis, tuberculosis of the lungs and of the mesenteric lymph-nodes. I have had a case complicated with tuberculosis of the mesenteric lymph-nodes.

The **symptoms** of diabetes in children, as given in the cases thus far published, do not extend over so great a period as in the adult. The cause of this must lie in the fact that there is a long period during which the symptoms are slight or escape notice. In a case which recently came under my care the child, nine years of age, showed symptoms only five months before she came under observation. At that time the mother noticed that the appetite was voracious and that there were great thirst and frequent urination. In spite of the large quantity of food and liquid taken, the child lost in weight. The amount of urine passed may be quite large. In Cotton's case it reached 104 ounces, in nine, 70 ounces daily. Monti has seen as much as 1½ litres passed in twenty-four hours. Eberth and Wirsching found that the daily excretion of sugar may be from 30 to 115 grammes to the litre.



In most of the cases recorded there has been polydipsia. The skin is the seat of a lichen-like eruption which causes intolerable itching. Furuncles and boils are also of common occurrence. The urine may contain albumin, and hyaline and granular casts. In my case albumin was present, but no casts. There is as a rule constipation. The temperature may be normal or subnormal. If there is complicating tuberculosis, there will be a slight daily rise of temperature toward evening. In all the cases thus far published there was progressive emaciation. Acetone in the odor of the breath and diabetic coma preceded by intervals of delirium close the clinical course of the disease.

The methods of diagnosis do not vary from those pursued in the adult. The urine of a child suffering from polyuria, polydipsia, a voracious appetite, pruritus, and progressive emaciation, should be carefully examined for sugar. Infants who take foods such as malted milk, containing an enormous quantity of sugar, often show a temporary glycosuria, which should not be mistaken for true diabetes, and which is not attended by any of the clinical symptoms of that disease (Epstein, Koplik).

### DIABETES INSIPIDUS.

(*Polyuria.*)

This is rare in infancy and childhood. If the daily amount of urine is three or more times the normal amount, there is polyuria. The specific gravity of the urine does not exceed 1006. Ebstein collected 10 cases in which the symptoms developed as a result of a cerebral inflammation in the vicinity of the fourth ventricle. The affection is sometimes hereditary. Cases have followed fright, the infectious diseases, meningitis, and traumatism. The cause is frequently obscure. The onset may be gradual or acute. Sometimes intense thirst or nervous symptoms usher in the disease. The nutrition may be maintained for years. The skin is dry, the body temperature below normal, and the symptoms do not differ from those manifested in the adult. The following case from my clinic was published by my assistant, Dr. Lewi:

Walter A., *et.* seven years, was first seen at the dispensary. The family history was, for the most part, negative, except that three children had died of nervous diseases, one of them, aged three years, of spinal meningitis, and two others, when babies, of convulsions. The patient when a baby was healthy; he was breast-fed one year and had never had a convulsion. When two years old he had varicella, followed by pertussis; at the age of five he had measles, complicated with an obstinate conjunctivitis, but recovered. In October, 1892, while driving, he was thrown from a carriage in rapid motion, striking the right side of the head; no ill effects were noticed

at the time. In January, 1893, he began to complain of pain in the back and in the nape of the neck. At about the same time it was noticed that he arose several times at night to urinate, and would invariably drink water after micturition; the mother noticed that he grew very nervous; the frequent micturition and increased thirst gradually became noticeable during the day, becoming so persistent that he was obliged to leave school. He was placed in a hospital, where he remained seven months; while there he lost flesh; none of the symptoms improved. He was on a rigorous milk diet during the entire time.

*Status Præsens.*—January 19, 1894, the child complains of pain on the right side of his head; says he feels chilly all the time and cannot stand still a moment. His face is pale and has an old person's look, with features sharp and pinched. The eyes are large and prominent, and the veins of the forehead dilated. The skin is exceedingly dry. The head is well shaped; careful palpation shows no sensitive spots. The chest is emaciated, with a slight rachitic girdle. The lungs, on auscultation give increase of voice-sounds at the right apex. The heart is normal, also the abdomen. The epiphyses of the ankles are enlarged. The glands at the angles of the jaw are enlarged, also those in the left axilla. *Urinary symptoms:* the child is passing a very large amount of urine; wakens on an average ten times a night to do so. The thirst varies with the amount of water passed; for the last few weeks he has complained of painful micturition. His appetite is excellent; he is on a milk diet. Weight is thirty-seven pounds; temperature (per mouth)  $97.8^{\circ}$  F. ( $36.5^{\circ}$  C.). The urine examination was as follows: quantity in twenty-four hours, 6400 c.c., colorless; specific gravity 1.003; reaction acid, no albumin, no sugar. Microscopical examination negative.

A series of quantitative urea tests were made in this case. The general consensus of opinion is that in cases of diabetes insipidus the amount of solids, including the urea, is increased. The tests were made with the Doremus ureometer. A control test was always made. The table shows marked diminution in the amount of urea. In order to avoid error, fresh bromine was used.

Date.	Sp. grav.	Amount in 24 hours.	Urea.
Jan. 25.	1.003	6300 c.c.	6.3 grammes.
Feb. 4.	1.003 $\frac{1}{2}$	6300 "	6.8 "
" 6.	1.005	5200 "	7.2 "
" 8.	1.002	7000 "	6.5 "
" 10.	1.004	5500 "	6.8 "
" 17.	1.002 $\frac{1}{2}$	7500 "	7.8 "
" 24.	1.003	6400 "	6.5 "
Mar. 18.	1.003	7000 "	8. "
" 30.	1.003	7300 "	7. "
Apr. 2.	1.003 $\frac{1}{2}$	6400 "	6.8 "

The treatment has been successful in some respects. The child was at once put on a general diet. Antipyrin was given. After the



first few days there seemed to be an abatement of the nervous symptoms and slight diminution in polydipsia, but no permanent improvement. He was then given opium several weeks without result. Ergot was next given, and continued for about two months; under this treatment the pain on the right side disappeared; the restlessness became less, and the thirst likewise diminished. Under a generous diet the child has held his own; he still weighs thirty-seven pounds, his color is healthy, and the mucous membranes are normal.

#### LEADING AUTHORITIES REFERRED TO IN CHAPTER IX.

- Bäumler, Ch.*: "Gelenk Rheumatismus," Deutsche. Klinik, Bd. ii., 1901.  
*Benecke*: Die gonorrhoeische Gelenkentzündung, Berlin, 1899.  
*Cotton*: "Diabetes in Childhood," Jour. Amer. Med. Assoc., 1901.  
*Charcot's works*, Arthritis Deformans, New Sydenham edit., vol. iii.  
*Hansemann, D. von*: Rhachitis des Schädels, Berlin, 1901.  
*Heubner, O.*: "Rhachitis u. Versuch mit Schilddrüsen Saft zu behandl.," Charité Annalen, xxi.  
*Jacobi, A.*: "Acute Rheumatism in Infancy and Childhood," Amer. Clin. Lectures, 1879.  
*Koplik, H.*: "Arthritis Deformans in a Child," Arch. of Ped., 1896.  
*Kassowitz, M.*: Rhachitis, i. and ii. Abtheil, Wien, 1882-5.  
 ——— Normale Ossification, Wien, 1881.  
 ——— Symptome der Rhachitis, Leipz., 1886.  
*Lewi, E.*: "Diabetes Insipidus," Arch. of Ped., 1894.  
*Miller, J. D. Milton*: "Acute Articular Rheumatism in Infants," Trans. Amer. Ped. Soc., vol. xi.  
*Moncorvo*: "Rheumatism Blennorrhagique Ches. les Enfants," La Méd. Infant, 1894.  
*Morse, J. L.*: "Rickets," Phila. Med. Jour., 1900.  
 ——— "Study of the Blood in Rickets," Medical and Surgical Report, Boston City Hospital, 1897.  
*West, J. P.*: "Rickets in Eastern Ohio," University Med. Mag., 1895.  
*Weil*: "Arthritis Deformans," Nouvelle Iconographique, 1890.  
*Zweifel, P.*: Aetiol. Prophylax. u. Therapie der Rhachitis, Leipzig, 1900.

## CHAPTER X.

### THE LYMPH-NODES, DUCTLESS GLANDS, AND DISEASES OF THE BLOOD.

#### THE LYMPH-NODES.

IN any disease or irritation of the scalp the nodes of the neck may be enlarged behind the border of the sternomastoid. The onset of some diseases of infancy, such as *rötheln* or rubella, is indicated by slow enlargement of these glands. Infection of the tonsils will cause the lymph-nodes at the angle of the jaw to enlarge and sometimes to suppurate. In young infants and children, chronic enlargement of the tonsils with adenoids causes an enlargement of these nodes. Tuberculous glands may occur in this region. The post-auricular lymph-nodes enlarge in disease of the ear or of the adjacent parts of the scalp. Parotitis will cause a sympathetic swelling of the lymph-nodes in front of the parotid, and also below this gland at the angle of the jaw and beneath it.

Retropharyngeal adenitis will cause the nodes behind the pharynx to swell and to appear at either side of the neck in front of the border of the sternomastoid muscle.

Any eruption on the chin will cause an enlargement of the lymph-nodes from the tip of the chin to the hyoid bone.

Swelling at the angle of the jaw will frequently simulate parotitis (Plate XXV.).

In certain forms of congenital syphilis with mucous patches on the lips and at the angles of the mouth (rhagades) there is beneath the body of the jaw a symmetrical enlargement of the lymph-nodes of both sides (syphilitic adenopathies). The lymph-nodes of the groin will enlarge in balanitis of the prepuce, syphilis, tuberculosis of ritual circumcision, and also in eczema and intertrigo of the inguinal folds. The lymph-nodes of the femoral region will in infants and children enlarge or suppurate as a result of any infection of the foot, leg, or thigh.

In the later stages of tuberculosis, either of the lung or peritoneum, there may be a general enlargement of the nodes of the neck, axilla, groin, and elsewhere. In many infants and children of a lymphatic diathesis (lymphatism), the nodes of the neck and groin show slight enlargement. Such enlargements should not, in the absence of positive signs of tuberculosis elsewhere, be hastily





Topography of Enlarged Lymph-nodes.

1. Preauricular enlarged, with disease of the external auditory canal, or any eruption on the face or parotitis.
2. Tonsillar nodes.
3. Submaxillary enlarged, with disease of the mouth, or skin eruptions over the lower jaw.
4. Submental enlarged, with chin eruptions.
5. Retropharyngeal enlarged, with infections of the pharynx and the retropharynx.
6. Nodes behind the border of the trapezius muscle enlarged, with disease of the scalp.
7. Nodes behind posterior border of the sternomastoid muscle enlarged, with infections of the retropharynx or the scalp.
8. Postauricular nodes enlarged, with mastoid disease or scalp infections.
9. Nodes above and behind the clavicle enlarged, with infections of the neck or mediastinum.
10. Nodes enlarged in infections of the hand or in eruptions such as those of syphilis.
11. Axillary enlarged, with infections of the arm, the axilla and the upper chest.
12. Nodes of the inguinal region enlarged in infections of the lower extremity, syphilitic or other lesions of the genitals.





pronounced tuberculous. After the exanthemata, the lymph-nodes of the neck, groin, and other regions may remain slightly enlarged. These enlargements usually retrograde to the normal in time, but if they remain rarely give rise to symptoms.

The physician should exclude every possible infection before concluding that an enlargement of the lymph-nodes in infancy and childhood is of a tuberculous nature. Cases of rachitis will show very slight enlargement of the lymph-nodes, especially in the inguinal regions. Forms of anæmia, such as von Jaksch's disease, also show these enlarged nodes. The lymph-nodes may be the seat of primary malignant disease, as in forms of lymphosarcomata. In malignant growths of the internal organs, such as the kidney, etc., they may be the seat of metastatic deposit. They are enlarged in acute and chronic forms of leukæmia. In these diseases the spleen and liver are also enlarged.

### ACUTE ADENITIS.

(*Acute Lymphadenitis.*)

The lymph-nodes in infants and children are peculiarly susceptible to acute infections, which are for the most part pyogenic (staphylococcic and streptococcic). Van Arsdale collected 500 cases of acute lymphadenitis seen in his experience. He found that 77 per cent. of them were in children. They are especially liable to the cervical infections. Eighty-five per cent. of the cases in children were infections of the lymph-nodes of the neck, the frequency in adults being only half as great.

**Etiology.**—Most of the infections of the lymph-nodes in children are, according to Van Arsdale, acute (79 per cent.). The majority of them are pyogenic. Children are subject to acute infections of the scalp, face, mouth, nose, tonsils, and mucous membrane of the nasopharynx. The lymph-nodes draining these regions are in the direct line of infection. Thus eczema and skin eruptions of all kinds, stomatitis of all varieties and inflammation of the tonsils and the nasopharyngeal space, will give rise to enlargement of the lymph-nodes. If the infection is severe, suppuration occurs. It is owing to these causes and to the breaches of surface caused by slight traumatism that this form of adenitis is so common. The essential exciting cause of acute lymphadenitis is the invasion of the nodes by pyogenic bacteria entering through the lymph-channels.

The **symptoms** of lymphadenitis in infants and children are essentially the same as in the adult subject. The node is at first felt as a hard nodular mass beneath the skin. One node or several may be infected. There is always some fever. At first the skin over the node is of normal color, but, as the inflammation progresses, it

becomes involved, red, and finally, if not treated, there will develop all the signs of an ordinary abscess.

The **diagnosis** is not difficult. The history and general course at once point to the nature of the disease. When the region about the parotid is affected, it is at times difficult to tell whether there is an infectious parotitis, or whether the nodes just beneath or above the parotid are involved. A preauricular gland situated in front of the ear on the parotid gland is apt to enlarge and suppurate. The nodes underneath the angle of the jaw and in front of the border of the mastoid sometimes enlarge and suppurate, involving the parotid by collateral swelling. In all of these cases, it is important to remember that a line drawn parallel to the lower border of the body of the jaw marks off the parotid above, and the lymph-nodes below. In exceptional cases, the swelling of infectious parotitis may extend lower than this line.

The **treatment** of acute lymphadenitis is at first abortive. Cold applications to the nodes which are enlarged and accessible, such as those of the neck, relieve the pain and in many cases lessen the severity of the reaction. This result is frequently seen in cases where infection of the nodes of the neck results from tonsillitis. Sometimes, in spite of all that can be done, suppuration occurs as a result of infection of cervical, axillary (vaccination), and inguinal nodes. In that case, the affected node should be incised. The further treatment of such cases is surgical.

### CHRONIC LYMPHADENITIS.

Chronic or subacute enlargement of the lymph-nodes in children may be pyogenic, tuberculous, or syphilitic. Of the cases collected by Van Arsdale, only 21 per cent. in infants and children were of chronic pyogenic origin, as against 12 per cent. in the adult. On the other hand, only 6 per cent. of all the cases of adenitis in infants and children were tuberculous. In the adult, the tuberculous forms of lymphadenitis are twice as frequent as in children. It is thus seen that even in chronic enlargements of the lymph-nodes of infants and children the occurrence of tuberculous forms gives the lowest percentage.

The **symptoms** of chronic enlargement of the lymph-nodes in infants and children are nodular tumors corresponding to the affected lymph-nodes. The enlargement may be single or multiple. Sometimes a whole packet of nodes is enlarged. The nodes most commonly enlarged are those at the angle of the jaw. This occurs in infants and children who suffer from chronically enlarged tonsils and adenoids. As a rule the nodes affected remain enlarged for months. At times they are somewhat less



swollen. They do not suppurate unless there is a tendency to a breaking-down of tissue. In all of these cases there is not only toxic irritation, but also a true hyperplasia of the tissue of the glands. I have seen these nodes removed and opened. Some of them have a soft, broken-down centre resembling that of the tuberculous nodes.

The **treatment** of chronic lymphadenitis is directed toward removing the source of infection. If the tonsils are enlarged and adenoids are present, they should be removed. A tonic course of treatment, good food, out-of-door exercise, iron, and cod-liver oil is indicated. In spite of these measures many cases do not improve. If the enlargement of the nodes in such cases is localized, the question of the advisability of removing them arises. That measure should not be resorted to unless there is a reasonable certainty that they are tuberculous, and when all other treatment has failed.

### DISEASES OF THE THYROID GLAND.

General enlargement of the thyroid is not uncommon in infancy and childhood. Normally the thyroid gland, and especially its isthmus, can be made out only by careful palpation. The isthmus

FIG. 156.



Enlarged thyroid in a child, six years old, who suffered from cardiac palpitation.

is indicated by a very slightly raised structure passing across the trachea beneath the cricoid cartilage. The lateral lobes cannot be

palpated. In cretinism and dwarfism, the enlarged lateral or supernumerary lobes beneath and just in front of the anterior border of the sternomastoid muscle can be palpated. Cystic growths of the thyroid are seen in front of the trachea, generally just above the notch of the sternum. They may occur in very young infants or in children of four or five years of age. Enlargement of the isthmus occurs chiefly in girls (Fig. 156). In these cases there is a disturbance of the heart functions and symptoms of the beginning of morbus Basedowii.

### CRETINISM, ENDEMIC AND SPORADIC.

Cretinism is a chronic affection which is characterized by a defective growth of the bones of the skeleton in their long axes, accompanied by a distinct set of mental symptoms and by changes in the soft parts.

**Forms.**—There are two forms, the endemic and the sporadic.

Endemic cretinism occurs in certain districts of Continental Europe. It does not exist in this country (Osler). The pictures presented by endemic and sporadic cretinism are similar. According to the recent studies of Dolega, His, and Bernard, their pathologic anatomy is also similar. Endemic cretinism is an advanced stage of a degeneration beginning with goitre manifestations. The resulting changes are due to "athyreosis," a suspension or disturbance of the functions of the thyroid gland. Sporadic cretinism, although also due to athyreosis, occurs without goitre. The peculiar formation of the skull in cretinism, endemic or sporadic, is now known not to be due to a premature synostosis of the os basilare and the sphenoid, as was at first thought by Virchow. The brachycephalic skull as manifested in a broadening of the bridge of the nose, and the prognathous expression is due to a deficient growth of the bones at the base of the skull, in their long axes. The sutures and fontanelles remain open for a long time. Dentition is delayed. The skin is myxœdematous in sporadic cretinism only. Dwarfism and anæmia are common to both forms.

#### Sporadic Cretinism.

**Occurrence.**—The disease may appear in utero or at any time after birth. Fully one-half of the cases develop before the eighteenth month (Fletcher Beach).

**Symptoms.**—I have published cases in which the symptoms appeared within a month or five weeks after birth. The history was as follows: In one case there was another cretin in the family; in the others there was no such history. The birth as a rule was normal (Fig. 157). The infant was jaundiced, but fairly well nourished.





PLATE XXVI.



Sporadic Cretinism. Child fifteen months of age.



It lay in a torpid state and was only roused when severely teased. The infant was easily chilled. The cry was deep and coarse. The forehead was low and narrow. The eyelids were puffy. The tongue was large, broad, and thick, at times protruding from the mouth. The abdomen was large, and the thighs and legs were out of proportion to the length of the trunk. The skin had a greenish hue. The thyroid gland could not be found. The surface was cool and the rectal temperature  $97^{\circ}$  or  $97.8^{\circ}$  F. ( $36.1^{\circ}$ – $36.5^{\circ}$  C.). The blood in these early cases has fetal characteristics. There is no

FIG. 157.



Congenital sporadic cretinism. Infant, four weeks old.

leucocytosis. In the cases which develop some months after birth the infant may at first be bright and normal. Six to nine months after birth, it may have some slight illness, such as an adenitis, and after this the change may be noticed, or the change may occur without any preceding illness. The infant ceases to notice objects about it, and becomes stupid and weaker. It may previously have attempted to walk or stand, but ceases to make an effort to do so (Plate XXVI.). The child's expression is idiotic. It has a meaningless smile most of the time and does not play. The skin has a wrinkled and myxœdematous appearance, the color being not

only pale, but also greenish. The nose is flattened, the lips are thickened, and the hair becomes dry and sparse. The forehead is

FIG. 158.



Sporadic cretinism; myxœdema marked. Child, twenty months of age.

narrow and the face has a prognathous expression—"monkey-like," as one mother expressed it. There are no teeth. The neck is short and thick. The genitals are large for the age. The skin



of the scrotum is thickened. The anæmia in these cases is extreme. The hæmoglobin may be as low as 18 per cent. (Fleischl). The leucocytes may be as high as 18,000, and the red blood-cells 5,600,000.

In other cases, the symptoms are at first more nearly of the myx-œdematous type. The skin, especially that of the face, has a greenish-yellow, waxy, puffy appearance. The upper and lower eyelids are swollen, as in nephritis. With these appearances, there are the dry hair, the macroglossia, the guttural voice, the dwarfish appearance, the protuberant abdomen, and the mental dulness. The expression of the face is less prognathous than in the first form. In one of my cases, the infant was in good health until the sixteenth month.

FIG. 159.



Cast of the hand of a boy cretin, four years of age. Flat and spade-like in form; it shows also the thickened and hypertrophied hypothenar eminence.

It then developed abscesses over the body, after which cretinism set in (Fig. 158). The abscesses were peculiar, the granulations slug-gish, and the pus was creamy. The skin was not œdematous but myxœdematous.

In both forms the hands are large, flat, and spade-like. The hypothenar eminence is thick, square, and hypertrophied, as in the lower animals (Koplik and Lichtenstein) (Fig. 159). In some cases the thyroid gland cannot be felt, in others it is small, and in excep-tional cases there is goitre (7 cases of Osler's series). In some of the older cases published, supraclavicular masses of fat or fatty tumors behind the sterno-mastoid muscles were found. I have failed to find them in the cases coming under my notice.

The **etiology** of sporadic cretinism is as yet absolutely unknown. Experimental and operative pathology have demonstrated that interference with the function of the thyroid gland (athyreosis) will produce a condition (myxœdema) closely resembling cretinism (Horsely, Reverdin, Kocher). The essential cause of endemic cretinism is thought to be some form of infection (Fagge). Sporadic cretinism is also ranked by some authors among the infections. Gull and Ord have described the form of myxœdema which develops spontaneously in adult life.

**Morbid Anatomy.**—There are cases of sporadic cretinism in which the thyroid gland is absent. It has not developed in foetal life and is not found at autopsy. In other cases there is found at autopsy a small atrophied gland which is sclerosed and much reduced in size. Such cases have been published as following the infectious diseases. Lastly, there are cases with goitre. The changes in the thyroid, when it is found in sporadic cretinism, have been described by Barker. There is an increase of connective tissue. The parenchyma is replaced by small and large irregularly shaped cells, which are granular and unlike the normal tissue. Some of the acini are almost solid; others are cystic and filled with colloid material. The cells may contain vacuoles; their nuclei may show "karyorrhexis." The nuclear changes are characteristic of degenerative processes. Some of the acini are replaced by connective tissue.

**The Bones.**—In the recent work of His, Dolega, and Bernard, it has been clearly shown that ossification in the pre-existent cartilaginous structures of the skeleton is delayed in all its phases. This is evinced in the delayed appearance of ossification centres, the delayed bony transformation of the epiphyses, and in the persistence of the epiphyseal zones. In some cretins, ossification is completed at a very late period of life; in others, infantile conditions are perpetuated. The dwarfing of the whole skeleton is thus explained, not by a premature synostosis, but by faulty proliferation and ossification of the epiphyseal cartilages. The bones of the skull are affected in the same manner as the vertebrae and the long bones, in that they fail to grow to their long diameters and in that ossification centres appear late.

The **diagnosis** is not difficult in advanced cases. The early cases require close study. In these, the stupidity increasing to absolute idioy, the retarded growth, the change in the expression, the swollen eyelids, thick lips, dry hair, wrinkled myxœdematous skin, the flat, spade-like hands, the dwarfish appearance, and the reduced internal temperature, all point to the diagnosis. In later cases, the extreme anemia, myxœdema, and pronounced pugnacious expression of the face are apparent.

Sporadic cretinism must be differentiated from the following conditions:





PLATE XXVII.



Mongolian Type of Idiocy. Child two years of age.



**Mongolian Idiocy.**—This is a form of genetous idiocy with which cretinism is frequently confounded. The idiots resemble cretins. The growth is stunted. The mouth is kept open. The tongue is large and fissured; the papillæ of the tongue are enlarged and erect. The tongue protrudes from the mouth (Plate XXVII.); the lips are thick; the voice is coarse and guttural. The temperature may be subnormal, but is generally normal. The skin is dry and the hair coarse. In young infants the skin may be delicate. The patients are easily chilled. The musculature is flabby. The infants cannot hold the head erect. The occiput is flattened, the neck short and thick. There is strabismus, and the axes of the eyelids have a Mongolian slant—that is to say, they converge. The inner eyelid comes down toward the nose with a rapid slope. The bridge of the nose is flat. The head is small and obtusely rounded; the antero-posterior diameter is nearly equal to the lateral one. The fontanelles remain open late. The skin, however, is not myxœdematous, nor is the expression prognathous as in the cretin. The anæmia is as a rule marked; in some cases the skin has a greenish hue. There is a curving inward of the tip of the little finger. The second phalanx is short and the terminal phalanx displaced. West has shown that although this deformity is very common in these idiots, it is not pathognomonic of Mongolian idiocy. Many of the subjects grow to adult life and have some degree of intelligence.

**The Dwarf with Idiocy.**—There may be several of these dwarfs in a family. The thyroid gland is enlarged at the beginning or during the course of the condition. The mental state is much stunted. The general growth of the body is retarded. The dwarfs are, however, well formed. The hands and extremities are perfect and the skin is not myxœdematous.

**Infantalism** combined with lipomatosis may be confounded with cretinism. In this form of disease there is no myxœdema and the skin is very delicate and soft. The genitals are atrophied. The expression of the face is that of child-like simplicity, the forehead is low and narrow. The hair is dry, and does not grow; the finger-nails do not grow. There may be, as in the case I published, blindness. The mental state is one of mild idiocy.

### Fœtal Rickets, or Achondroplasia.

(*Chondrodystrophia Fœtalis.*)

Cases have been reported in this country by Jacobi, Smith, and Townsend. Thomson, of England, has described the affection as of intra-uterine origin. Although Horsley and Barlow classify the cases with sporadic cretinism, they should, for clinical purposes, be regarded as a separate set. They have nothing in common with rachitis or cretinism (Thomson). The patients are far

from being idiotic or presenting any of the symptoms of myxœdema. Some of the dwarfs who reach adult age are exceedingly clever. Many of them are performers in museums and are above the average in intelligence. The case published by Townsend was that of a stillborn infant.

Parrot and Jacobi have described infantile cases. The affection is due to absence, arrest, or perversion of the normal process of endochondral ossification in every bone in which it takes place in utero.

The **treatment** of cretinism constitutes one of the most marvellous chapters of modern medicine. The administration of thyroid extract results in a partial restoration of the mental capacity and a return to growth and development approaching the normal. I published in 1897 some cases of cretinism diagnosed early in infancy, in which the treatment was begun at once. In those in which the treatment was begun at the age of one month, the children have become bright and apparently normal. In those in which it was inaugurated at the fifteenth month, the children have, after five years of treatment, remained somewhat backward in mental development. One patient, now a boy of six years, goes to school and recites his alphabet, but is very simple in manner. In these late cases the treatment does not give the complete results at first expected.

I begin with the dried extract, grain  $\frac{1}{2}$  (0.03) t. i. d., and increase the dose until the infant takes a grain j (0.06) three times daily. After the symptoms have retrograded, the dosage is kept stationary for a few months. It is then reduced or the remedy is given only every other day. If symptoms, such as stupidity, pallor, or reduced temperature, reappear, the dose is increased. The first sign of improvement is a reduction of the anæmia, as evidenced in the increase of hæmoglobin. The body temperature rises to the normal. The skin becomes of normal delicacy and supple. The stature increases and the hair becomes glossy. Thomson, of Edinburgh, has published cases of adult cretins whose bones became softened after the prolonged administration of thyroids. These were cases in which treatment was begun late in life. The symptoms of excessive administration of thyroids include rise of temperature and slight diarrhoea.

I have found thyroid therapy of doubtful utility in cases of Mongolian idiocy. In the dwarfs above mentioned, it causes increase of stature; the intelligence, however, remains backward.

### THE THYMUS GLAND.

**Landmarks.**—The thymus is a glandular organ enclosed in a capsule. It is situated in the anterior mediastinum, and contains a white tenacious fluid substance which is present in varying quanti-



ties. Sappey shows that the thymus in the newborn infant extends from the upper edge of the manubrium sterni, 5 cm. downward. Its upper border may reach the isthmus of the thyroid or may be removed  $2\frac{1}{2}$  cm. from it. It extends downward to the middle or upper third of the pericardium. In exceptional cases it may have a longitudinal diameter of  $11\frac{1}{2}$  cm., reaching the diaphragm (Triesethau). The thymus is about 2 to 3 cm. wide. Luschka makes it unsymmetrical, consisting of two lobes united by an isthmus. It lies over the course of the pulmonary artery and is surrounded by a reflection of the pericardium. It is separated from the sternum by loose connective tissue. Its length varies from 4 cm. in the nursing, to 11 cm. in the ninth year, the average ratio to the body length being 1 to 7 or 8.

**Weight.**—Its weight varies. In the results which I obtained in collaboration with Jacobi, it did so within wide limits. In infancy the average weight is 20 grammes; from the second to the fourteenth year it is 24 grammes. After the twenty-fifth year the thymus atrophies and may weigh 2.2 grammes (Friedeleben). In abnormal states the weight may be 32 grammes (Triesethau, Pott). The causes of the enlargement of the gland and the conditions under which it occurs are not as yet known. The gland is large in infants dying of the most diverse diseases.

**Percussion.**—Under the most favorable conditions it is difficult to ascertain the exact size. The thymus has sometimes been marked out as large during life, and post mortem found to be small. As a rule, an area of dullness situated behind the upper part of the sternum, and discernible on gentle percussion, may be cautiously interpreted as due to the thymus (Sahli). An unsymmetrical area giving dullness on one side of the sternum is probably due to the thymus (Luschka), especially in subjects under the second year.

**Abnormal Conditions.**—None of the abnormal conditions of the thymus can be diagnosed with certainty during life.

### Hypertrophy of the Thymus.

Hypertrophy of the thymus has been observed by Virchow, Grawitz, Pott, Jacobi, and others. The symptoms caused by the exceptional enlargement have been grouped by Virchow, Grawitz, West, and Goodhardt as a symptom-complex which is described in the literature under the heading of "Thymic Asthma." Many cases are attended by a form of laryngismus stridulus and difficult breathing, and eventuate in convulsions and sudden death. There has been much discussion as to the existence of thymic asthma. It has been doubted that sudden death can be caused by a large thymus. The conclusion of Virchow, Grawitz, Pott, and others, that it may do so, must be accepted. In the case of Pott the thymus weighed 32

grammes, was 9 cm. long and  $1\frac{1}{2}$  cm. thick. By compressing the upper air-passages, the large venous vessels, the right ventricle, or the recurrent nerves, an enlarged thymus may cause sudden death. That possibility is denied by Friedeleben. In the work of Jacobi, it was shown that hemorrhages of the thymus are not uncommon, and are present in a number of conditions, especially in pertussis. Inflammation of the thymus may be present in inflammatory conditions of the pleura and pericardium. Steudener has published a case of sarcoma of the thymus, and Vogel one of carcinoma of that organ, occurring in childhood. Demme published a case of isolated tuberculosis of the thymus. In the monograph of Jacobi, general tuberculous infection of the thymus was investigated, as was also the condition as found in diphtheria. In the latter disease, I have found the same condition of necrobiosis of the thymus described by Oertel as occurring in other organs. Congenital syphilis may manifest itself in arterial and connective-tissue changes. Abscess of the thymus is rare.

### THE SPLEEN.

**Anatomical.**—At different periods of childhood the length of the spleen varies from 4 to 10 cm., the breadth from 2 to 5 cm., the average thickness being about 0.5 cm. It forms an oval-shaped body, behind the ninth, tenth, and eleventh ribs, the long axis running in the direction of the ribs. Up to the second month of life, the anterior edge of the spleen is found in the midaxillary line; after that, it may be found further forward than this line, or posteriorly to it. The upper edge corresponds to the upper edge of the ninth rib; the lower border to the lower border of the eleventh rib. The spleen may be located by percussion and palpation.

**Percussion.**—The patient is caused to lie on the back. It is not necessary to cause children to lie in an inclined lateral posture. The upper border is first located by percussing from above downward in the midaxillary line on the left side. At the seventh rib is a strip of slight dulness extending from the seventh to the ninth rib (Fig. 95). I have been able to locate it in infants and in children under the age of six years. There can be no question as to its existence, although there may be doubt as to its causation. Symington, in his frozen section, shows that, in a girl six years of age, the left lobe of the liver is distinctly on the left side behind the seventh and ninth ribs. Sahli ascribes the strip to what he calls the deep dulness of the spleen. From the ninth rib downward, there is absolute dulness, then flatness, due to the presence of the spleen proper behind the chest wall. The anterior border of the spleen is located by percussing in a horizontal direction toward the axillary line along the tenth rib.



**Palpation.**—The enlarged spleen can be distinctly made out by palpation. The abdomen should be relaxed. It is sometimes necessary to flex the thighs slightly, in order to relax the abdomen. In young infants it is not necessary.

The physician stands at the *right side* of the patient and with the palmar surface of the fingers of the right hand palpates the abdominal parietes just beneath the border of the ribs (Fig. 160). As the patient inspires deeply, the hand is by steady pressure insinuated beneath the ribs in an upward and backward direction. In the vast majority of cases under the tenth year, the normal spleen may thus be felt.

In practice, it may safely be said that a spleen which cannot be felt below the border of the ribs is not enlarged, unless some condition, such as the presence of fluid or tympanites, prevents thorough palpation. I have rarely failed to palpate the enlarged spleen

FIG. 160.



Method of palpating the spleen.

satisfactorily. Enlargement of the spleen is found in rachitis, chronic gastro-enteritis, sepsis, typhoid fever, malarial fever, varicella, syphilis, anaemia infantum pseudoleukæmica, leukaemia, congenital syphilis, cirrhosis of the liver, amyloid degeneration, heart disease, and simple catarrhal jaundice.

From these statements it will be seen that enlargement of the spleen in infancy and childhood is pathognomonic of no one disease, and should not lead to any one conclusion. It is only corroborative in the presence of other signs and symptoms. Without a very thorough and painstaking examination of the blood, the significance of the enlarged spleen in the febrile and afebrile affections cannot be determined. In enlargements of the spleen such as are met in rachitis, heart disease, syphilis, chronic gastro-enteritis, icterus, varicella, examination of the blood may not be necessary.

### Splenic and Kidney Tumors.

In rare cases in which sarcoma of the left kidney is suspected, it may be necessary to exclude tumor of the spleen.

An enlarged spleen is smooth on the surface and has a sharp anterior edge interrupted by an indentation—the hilus. The tumor is pointed and sharp below. It can be grasped deep in the lumbar region behind.

Kidney tumors are irregular on the surface and marked out into lobes, some of which may be cystic. The tumor projects upward behind into the lower part of the chest. The whole lumbar region is flat on percussion. The borders of the tumor are rounded. On the other hand, I have made an autopsy in a case of cirrhosis of the liver and spleen in which the latter organ during life showed uneven tumors on its surface (*gummata*). The physician must be partly guided by the history of a case. The urine should be examined in cases of sarcoma of the kidney, and the blood in cases of enlarged spleen. I have seen a subphrenic abscess displace the spleen downward. The left lobe of the liver was also displaced in the same direction. Under anaesthesia, a round mass could be felt above the spleen, which was enlarged. Behind, the lung came well down to the bottom of the chest, as was evinced by the presence of the respiratory murmur. Dulness was, however, present in the left axillary line and behind. On exploratory puncture in the posterior axillary line, the subphrenic abscess was found to be present.

## THE BLOOD.

### LEADING GENERAL CHARACTERISTICS OF THE BLOOD IN INFANCY AND CHILDHOOD.

For diagnostic purposes, it is important to bear in mind certain characteristics of the blood in infancy and childhood. Ehrlich has shown that conditions normal to the blood in early life are of grave import if found in the adult.

**The Red Blood-cells—the Erythrocytes.**—During the first three days of life, nucleated red blood-cells are found in the normal blood. In the newly born infant, the red blood-cells number from 4,500,000 to 6,500,000 to the cubic millimetre (Hayem). There is a polycythæmia. This condition is found during the first few days of life. On the fourteenth day there is an average of 5,500,000 red blood-cells to the cubic millimetre. From the second to the tenth year the average number is 5,000,000 (Otto, Schiff, Sörenson). The polycythæmia in the newly born infant is greater if the tying of the umbilical cord is delayed until its pulsations cease. Weaklings



show a diminished number of red blood-cells. In addition to imperfect nutrition, anæmia of any kind, acute or chronic cachexia, and certain drugs, such as antipyrin, antifebrin, phenacetin, and lactophenin, reduce the number of red blood-cells by disintegrating a certain proportion of them (Monti). Infectious diseases, such as malaria, scarlet fever, typhoid fever, and sepsis, have a similar influence. In severe anæmia, such as that accompanying rachitis, nucleated red blood-cells appear in the blood. These are also found in the severe primary anæmias, in acute leukæmia, and in pernicious anæmia of infants and children.

**The White Blood-cells—the Leucocytes.**—The number of leucocytes in the newly born infant is high, being from 18,000 to 30,000 to the cubic millimetre (Hayem, Guppen). It gradually falls to 12,000 to the cubic millimetre, the average for infants. The percentage of lymphocytes is at first small in comparison with that of the polynuclear leucocytes. Gundobin, whose work has been confirmed by Carstanjen, found that the polynuclear leucocytes preponderate in the newborn infant. They increase and reach their highest figure in the first forty-eight hours of life. They then diminish in number, while the mononuclear lymphocytes increase proportionately until the seventh or tenth day, when the blood assumes the characteristics which distinguish it during the period of infancy. During infancy the mononuclear lymphocytes are more numerous than the polymorphonuclear leucocytes. The following table is taken from Gundobin's figures :

	Polymorpho- nuclear leucocytes.	Mononuclear lymphocytes.	Transitional forms.
Immediately after birth . . . . .	63 per cent.	25 per cent.	12 per cent.
Forty-eight hours after birth . . . . .	70 “	21 “	19 “
Infancy . . . . .	34.6 “	59 “	6.4 “

In normal infants and young children, the number of leucocytes to the cubic millimetre may vary from 13,000 to 20,000 (Japha). The so-called digestive leucocytosis found in the adult is inconstant in infants and young children (Japha). There is undoubtedly an inflammatory leucocytosis in infants and children similar to that seen in the adult. It occurs in pneumonia, measles, scarlet fever, rheumatism, sepsis, diphtheria, post-hemorrhagic anæmia, and cachexia (sarcoma). In the normal state, the leucocytes may reach a minimum of 6000 to the cubic millimetre (Monti). This fact should be borne in mind in estimating the leucopenia in typhoid fever, malaria, tuberculosis, and in other infectious or toxic states.

The transitional forms of leucocytes are numerous in the newly born infant, reaching their maximum from the sixth to the ninth day. The eosinophiles are present in the same number as in later life (Japha).

**The Hæmoglobin.**—The blood is richer in hæmoglobin at birth

than later in life (Morse, Leichtenstern, Rotch). After birth the percentage of hæmoglobin sinks, and at the third month reaches that of later life. Carstanjen found the hæmoglobin to reach on the average 100 per cent. up to the twelfth day. The lowest percentages are found from the sixth month to the second year. There is, in exceptional cases in normal children, a very high percentage from the fifth to the tenth year, ranging from 95 to 110 (Widowitz, Leichtenstern, Hock, and Schlessinger). The percentage in healthy children may be as low as 60 (Fleischl) or 8.4 grammes to 100 c.c. of blood. At the third month of infancy it may range from 69 to 94; up to the second year it may range from 62 to 81 (Monti). There seems to be no fixed normal limit. Anæmia or toxæmia of any kind and infectious diseases diminish the hæmoglobin.

**The Specific Gravity.**—The exact clinical significance of the specific gravity of the blood is little understood. The specific gravity is high in the newly born infant, ranging from 1.056 to 1.066. From the sixth month to the tenth year it varies from 1.050 to 1.056 (Monti). These figures correspond to those of Hock, Schlessinger, Lloyd, Jones, and others. The blood of strong children and breast-fed infants has a higher specific gravity. Diarrhoea may raise it, but rarely to a ratio of more than 0.004 part per 1000. The specific gravity is increased in the infectious diseases, pneumonia, pleuritis, endocarditis, typhoid fever, and tuberculous meningitis, and falls on the decline of these processes. It is also increased in congenital heart disease, chorea with endocarditis, icterus, and diphtheria. It diminishes with the loss in weight accompanying anæmia and nephritis, and in cachexia (Hock, Schlessinger, Monti, Hammersley, and Felsenthal).

### ANÆMIA.

Anæmia is a condition resulting from a deficiency in the blood of one or more of its constituent elements. It may be either congenital or acquired. In the latter case it may either be secondary to other conditions or occur as a primary disease. Congenital anæmia is seen at birth in infants born of badly nourished mothers, who during pregnancy have suffered from some disease of the placenta, or from syphilis, tuberculosis, or malaria. The fœtus in utero becomes anæmic. Acquired anæmia appears after birth. It is either secondary to some acute loss of blood (post-hemorrhagic), to chronic loss of blood, or is caused by defective nutrition, unhygienic surroundings, diseases of the various organs, toxæmia, infectious diseases, or parasites.

Primary or essential anæmia is the form in which the changes in the blood play so important a rôle that it is assumed there is a dis-



ease of the blood itself or of the blood-forming organs (Monti). Such are the forms of leukæmia, chlorosis, and pernicious anæmia.

### Simple Anæmia.

(*Secondary Anæmia.*)

Secondary simple anæmia may follow some acute or chronic loss of blood. In acute post-hemorrhagic anæmia, the increase of fluid elements keeps pace with the loss of blood if the loss, though small, is repeated at short intervals. Children show the effects of loss of blood much more quickly than adults. Hydræmia is the condition which results when the loss is marked. The fluid elements increase, and there is a diminution in the specific gravity of the blood and in the amount of hæmoglobin. Hydræmia may result in children without hemorrhage; that is to say, it may occur in extreme severe anæmia secondary to some disturbance of nutrition or to illness. In post-hemorrhagic anæmia the coagulability of the blood is increased immediately after the hemorrhage. Ehrlich supposes this to be due to an increase in the number of blood-plates. After the hemorrhage, the regeneration of blood is in the infant, as in the adult, indicated by the formation or appearance in the blood of microcytes, megalo-cytes, and nucleated red blood-cells (normoblasts). The severe forms of this variety of anæmia also show polychromatophilic properties of the red blood-cells. These are so poor in hæmoglobin that with various stains the normal reaction is very much changed. There are various shades of the stained red blood-cells. In recent and severe cases of post-hemorrhagic anæmia there may be leucocytosis. There is an increase of the polynuclear neutrophilic leucocytes (Monti, Ehrlich). Nucleated red blood-cells (normoblasts) may appear in severe cases. Poikilocytosis is also one of the changes seen in the blood.

Secondary anæmia of a mild or of a severe type is also seen in infants and children who suffer from defective nutrition. It complicates or accompanies rachitis, syphilis, scrofula, tuberculosis, gastro-intestinal catarrh, chronic endocarditis, purpura, morbus Werlhofii, and infectious diseases.

The **symptoms** of mild anæmia in infants and children do not differ materially from those of adults. The patient is pale and the mucous membranes have a characteristic pallor. The appetite is capricious. The patients also suffer from symptoms due to the primary affection—syphilis, rachitis, acute infectious disease, gastro-enteric disturbance (acute or chronic), or cardiac affection. The pallor of cardiac disease or nephritis is characteristic in infants and children, as in the adult.

The anæmia if of a severe type takes the hydræmic form. In the severer forms of anæmia, especially in infants and very young

children who suffer from syphilis or rachitis, the skin is waxy white or yellowish. The ears are absolutely devoid of any color of blood. In cretinism the skin has a greenish-yellow hue. Infants do not show the symptoms, such as dyspnoea or palpitation, seen in older children on exertion. The muscles are flabby and there is a disposition to lie quietly in the crib. The spleen may be large, and the liver also, especially if rachitis or syphilis is present. In cases in which the anæmia is extreme, the spleen may be normal.

Infants and very young children do not always show the anæmic murmurs which are heard over the heart area in older children. In older children murmurs of that variety may be present with a venous hum in the neck, and the symptoms of mild and severe anæmia are essentially those of later life. These are indisposition to exertion, feelings of weakness, drowsiness, lack of appetite, irritability, and restlessness. Some of the severe forms of anæmia show for weeks a very slight irregular febrile curve. In many cases the fever is due to intestinal toxæmia.

**The Blood.**—The mild forms of simple anæmia may show only a diminution in the amount of hæmoglobin, a very slight diminution in the number of red cells, a reduction of the specific gravity, and if there is a primary affection which, like pneumonia, causes an increase in the number of leucocytes, leucocytosis. My records of severe forms of anæmia in infants and young children show a diminution in the amount of hæmoglobin (18 per cent.). The blood shows microcytes, megalocytes, megaloblasts, and normoblasts. Increase of mononuclear lymphocytes is proportionate to that of the polynuclear leucocytes. Poikilocytosis in various forms is present, as are also polychromatophilic phenomena. In the severe forms of anæmia due to malarial poisoning I found, in addition to the plasmodium, microcytes, megalocytes, and megaloblasts. The eosinophiles are not increased. In severe anæmia, the physical characteristics of the blood are striking. It may be so thin as to separate on puncture into a reddish and a colorless portion resembling beef-water.

### Chlorosis.

Chlorosis is a form of primary anæmia. It is not a disease of infancy or childhood, and is mentioned here only in order to complete the classification of diseases of the blood. Its etiology is obscure. Virchow believed it to be due to congenital narrowness of the whole arterial system and smallness of the heart. This theory does not explain the cases in which recovery takes place. Meinert ascribed the condition to an irritation of the abdominal sympathetic. Hofman thought that developmental conditions of the genital apparatus were causal in chlorosis. Foreheimer contends that intestinal



auto-infection is etiological in producing the chlorotic state, since there is in chlorosis an interference with the production of hæmoglobin, the principal source of which is the gut.

**Occurrence.**—Chlorosis is more common in females than in males, and occurs at the time of puberty.

The condition of the blood has been described by Monti. The hæmoglobin is diminished. The number of red blood-cells is in mild cases scarcely at all reduced. In severe cases it may fall to 1,000,000 to the cubic millimetre. The absolute amount of hæmoglobin may reach 4 to 8 in 100 cubic millimetres of blood. The specific weight may be reduced to 1035. There are microcytes in the blood. There is no leucocytosis. There are poikilocytosis and polychromatophilic appearances in the stained blood.

### Pseudoleukæmic Anæmia of von Jaksch.

(*Anæmia Infantum Pseudoleukæmica.*)

In 1889 von Jaksch described a symptom-complex met with among infants and young children, to which he gave the name of anæmia infantum pseudoleukæmica. He described the condition as a clinical entity which, in running its course, gives the picture of severe lymphatic anæmia. There are enormous enlargement of the spleen, slight enlargement of the liver, some enlargement of the lymph-nodes, and changes in the blood. It is a secondary anæmia rather than a distinct disease. For this reason Fischl, Epstein, and others deny that it is a clinical entity. On the other hand, Monti and Luzet have described numbers of cases. I have records of 5 cases, 1 of which was published. The anæmia is extreme.

**Etiology.**—It is difficult to determine the etiology. Von Jaksch and Monti trace an intimate connection between this condition and rachitis. Wentworth and others regard it as secondary to some form of intestinal infection.

**Occurrence.**—The condition is rarely found before the age of six months. My cases ranged from the ages of twelve to eighteen months. It may occur up to the third year, and is most common from the seventh to the twelfth month. Most of the cases thus far published have occurred in infants or children suffering from rachitis or congenital syphilis. In all of my cases there were signs of rachitis. Some of the children had previously suffered from chronic gastro-enteric derangement.

**Morbid Anatomy.**—The spleen is large and fills the left hypochondrium, sometimes reaching the crest of the ilium. It is hard, smooth, and has a sharp border. There is an increase of the cellular elements. A small number of hæmoglobin-bearing cells of the size of red blood-cells are found.

The liver is slightly larger than is normal, but is of normal

consistency and color. The lobuli are less distinct than is normal. Luzet has described cellular elements in the liver containing finely granular protoplasm. These are not like liver cells. They have small sharply defined nuclei, sacculated in shape. They are surmised to be forerunners of the red blood-cells. The liver cells are normal. The lymph-nodes are only moderately enlarged, and do not form lymphomata, as is the case in leukæmia. There are no changes in the bone-marrow.

**Symptoms.**—The infants affected have as a rule suffered from chronic intestinal disturbances. Most of them are bottle-fed and atrophic. Although the skin is intensely anæmic and of a yellow,

FIG. 161.



Pseudoleukæmic anemia, enlarged spleen and liver.

waxy tinge, there is sometimes a panniculus of fat. The musculature is flabby and the abdomen large. As a rule there are signs of rachitis. The fontanelle is open and the eruption of the teeth delayed. The infants are irritable, peevish, do not willingly take food, and do not assimilate it. In one of my cases, there was complicating pneumonia.

There is, as a rule, no fever, unless it is due to intestinal toxæmia. The picture is one of progressive emaciation and anæmia. In some cases there is complicating icterus, and the spleen reaches to the crest of the ileum. The edge of the spleen is sharp and the hilus can be distinctly felt. The liver is slightly enlarged; its edge is round and



smooth. In one of my cases, it extended two and one-half inches below the free border of the ribs (Fig. 161). The lymph-nodes in the groin and axillæ are slightly enlarged, sometimes only to the size of a bean.

**The Blood.**—The specific gravity of the blood is reduced. The hæmoglobin may be reduced to one-quarter the normal percentage. It may be as low as 17 per cent. (Fleischl). There is a marked diminution of the number of red blood-cells. The nucleated forms of erythrocytes are abundant. There are megaloblasts, which show karyokinesis. In addition there are red blood-cells of all sizes—microcytes and megalocytes. There is poikilocytosis to a marked degree, and also polychromatophilia. The leucocytes are only moderately increased. In the severe cases the proportion of white blood-cells to the red may be as 1:100, 1:80, or 1:15 (Monti). The picture given by the leucocytes is different from that of leukæmia. Most authors agree that the various forms are represented and increased in equal ratio. In my cases, the mononuclear lymphocytes, as well as the transitional forms, were increased.

The **course** of the disease is chronic. In most cases, the children succumb to progressive weakness and emaciation, or to intercurrent disease. Recovery may take place. I have seen one case of aggravated form improve under treatment. Whether these cases can, as stated by Monti, pass into a leukæmia or pernicious anæmia is a matter of doubt.

**Treatment.**—Thus far the treatment has been empirical. Small doses of Fowler's solution are indicated. If rachitis is present, phosphorus is given by some in small doses. I have seen cases do badly under that treatment. Tonics and an easily assimilable diet are indicated. The bowels should be kept clear by enemata given daily in order to lessen the possibility of infection of the gut.

### Leukæmia (Leukocythæmia).

Leukæmia is a persistent condition of the blood in which there is an increase of the white blood-cells, and a diminution of the red ones. It is a primary disease of the blood itself. Accompanying it, there are changes in the spleen, liver, bone-marrow, lymph-nodes, and lymphoid tissues. Virchow called the condition "white blood." French writers have called it leukocythæmia. The proportion between the white and the red blood-cells is not so distinguishing a feature as the appearance of large numbers of lymphocytes in the blood, in which they are normally present in only small numbers. In one form the appearance of mononuclear neutrophile-staining myelocytes which are normally absent is a distinguishing feature. Ehrlich characterizes leukæmia as a mixed leucocytosis of chronic course, since white blood-cells of all kinds are present in

the blood. This is not the case in the polynuclear and eosinophile leucocytosis.

**Occurrence.**—The disease is rare in childhood, but some authors believe it to be more common in the first year of life than is generally supposed (Monti, Mosler). Fifteen to 20 per cent. of the cases occur in the first decade of life (Baginsky). Males are more frequently affected than females. The disease is believed to be hereditary.

The **etiology** of the affection is still unknown. In a few cases, micro-organisms and sporozoa have been found in the blood (Roux, Kelsch, Veillard, Lowit). The sporozoa of Lowit are described by him as being free in the blood as well as in the leucocytes and in the blood-making organs. In lymphatic leukæmia they are described as being intracellular only.

Some writers think that ræchitis and syphilis predispose to the development of leukæmia, especially if the bones are involved as well as the liver, spleen, and lymph-nodes. Certain forms of anæmia following malaria, diphtheria, and scarlet fever, and accompanied by enlargement of the liver, spleen, and lymph-nodes, may, according to some writers, pave the way for leukæmia. Physical or mental strain, unhygienic living, defective nutrition, and traumatism of the spleen, have all been regarded as predisposing factors.

**Forms.**—The simplest classification of leukæmia is that based upon the anatomical appearances of the blood. Such is the classification of Ehrlich, which is as follows :

(a) Lymphatic leukæmia, in which there is a marked hyperplasia of lymphoid tissue.

(b) Myelogenous leukæmia, in which there is hyperplasia of myelogenous tissue. Lymphatic leukæmia may run an acute or a chronic course. In both forms the distinguishing feature is the appearance in the blood of large numbers of the mononuclear lymphocytes and the displacement of the polynuclear leucocytes. The acute form is rare. It occurs in childhood. Four cases have occurred in my hospital service in the past two years. Its course is rapid. There are slight or marked tumor of the spleen, slight or very marked enlargement of the liver, and a tendency to petechiæ and to general hemorrhages. Some authors regard these cases as infectious. The chronic forms show marked enlargement of the spleen.

**Changes in the Blood.**—As was previously stated, the lymphatic forms of leukæmia are distinguished by the appearance, in the blood, of large numbers of the small and large mononuclear lymphocytes. In the myelogenous forms, a cell which is normally not present in the blood, but is indigenous to the bone-marrow, appears in large numbers. This cell is the large mononuclear neutrophilic staining cell, the myelocyte of Ehrlich. The mast-cells are also



found in these cases, but are not peculiar to this form of anæmia. In addition there is in the myelogenous forms of leukæmia an increase in the number of all three types of granulated white cells, the neutrophiles, the eosinophiles, and the mast-cells. There are dwarf forms of the white blood-cells, mitoses, and lastly large numbers of nucleated red blood-cells. Normoblasts, megaloblasts, and myelocytes are not normally present in the blood. They are occasionally found in pneumonia, and in leucocytosis. The eosinophiles are increased to fifteen times their normal number. The slow coagulability of leukæmic blood is characteristic.

The spleen is enlarged. It is at first soft, often firm, and is infiltrated with lymph-cells. The capsule is thickened; the connective-tissue stroma is increased and infiltrated with lymph-cells. The lymph-nodes show similar changes, and may be enlarged, forming tumors of considerable size.

The bone-marrow is so infiltrated with lymph-cells as to acquire the appearance of a purulent infiltration. The same lymphoid infiltration is found in the liver. The follicles of the gut are swollen. There is an increase of lymphoid cells and tissues. The lymphoid tissues elsewhere, such as the tonsils, thymus, skin, and even the retina, show the same changes. There are hemorrhages and exudate in the ear, and the nerves and nervous tissue of the central nervous system are the seat of lymphoid cellular invasion.

**Symptoms.**—**Acute Leukæmia.**—Cases of acute leukæmia in infancy and childhood have lately been increasing in the literature. The most recent cases include those of McCrae, in a boy aged three years, and of Miller, in an infant of eight months. Cases have also been reported by Morse, Japha, Strauss, Monti, Berggrün. The symptoms in all the published cases were similar. In a boy eight years old, admitted to my hospital service, there were no premonitory symptoms. Two months before admission he was in good health. He became very pale, there were irritability and loss of appetite, and the abdomen increased markedly in size. He complained of pains in the legs, and at the onset had chills and fever every other day. After the appearance of the chills he suffered from a low irregular fever. A week before death, the skin had a waxy color, there were petechiæ on the extremities, the gums bled easily, and the lymph-nodes of the axillæ and groin were enlarged. There was an anæmic murmur with the first sound of the heart; the liver was enlarged below the free border of the ribs to the extent of two fingers' breadth; the spleen was enlarged to the level of the umbilicus; the fundus of the eye showed retinal hemorrhages. Examination of the blood showed the hæmoglobin to be reduced to 15 per cent. (Fleischl). The red blood-cells numbered 1,012,000 to the cubic millimetre; the white blood-cells, 37,000. There was an immense preponderance of lymphocytes (mononuclear). The patient died with

signs of progressive weakness. Coma was preceded by vomiting and the appearance of a few petechiæ. The blood state continued much the same as at first. In another case the number of mononuclear lymphocytes was fully 75 per cent. of the white blood-cells. In both these cases the spleen and liver diminished before death. The proportion of white to red blood-cells may not be far from normal. In another case the nucleated red blood-cells, large and small, were very numerous. In this case, in a boy of five years, the nodes around the parotid and angle of the jaw, in the axilla, and in the inguinal region, increased in a short time to a large size, and the spleen grew rapidly larger and reached to the crest of the ilium. The liver reached to the umbilicus. These mediastinal lymph-nodes were enlarged and caused great dyspnœa. The distress was very great just before the lethal issue. In a case of v. Noorden's the proportion of the white to the red blood-cells was 1:200. The character of the white blood-cells is diagnostic. Most of the cases published showed a slight temperature. The fatal issue usually results a few weeks or a month or two after the onset of symptoms.

**The Chronic Form.**—The symptoms of the chronic form extend over a greater length of time. For months there are anæmia, lassitude, and extreme physical weakness. The appetite is good, but in spite of abundant nourishment, emaciation is progressive. In some cases there are periodic diarrhoeal attacks. Profuse hemorrhage may occur without warning either from the nose or intestines. Chills and fever resembling those of paludism are sometimes present. None of these symptoms is particularly characteristic. As the disease progresses there are headache and pain in the limbs and in the region of the spleen. The anæmia after a time assumes a severe type, and the skin becomes waxy and yellow. At this stage the spleen and liver enlarge and distend the abdomen. There are dyspnœa and palpitation; the anæmia takes the hydræmic form, and there is œdema of the face, hands, and feet. Hemorrhages then occur from the nose, lungs, mouth, intestines, but rarely from the kidneys. There are petechiæ in the skin and hemorrhages in the retina. In the lymphatic form the lymph-nodes in various parts of the body enlarge and form masses which are painless and covered with unaffected skin. The skin may be affected by the process. The mesenteric nodes may sometimes be felt through the abdomen. The spleen attains an enormous size. The liver may extend as far as the umbilicus. Respiratory difficulties, heart weakness, and nervous symptoms (such as vertigo, somnolence, and coma) end the clinical course of the disease. The urine is diminished, and contains hyaline casts, lymphoid cells, and a trace of albumin. There may be a slight continued fever in the course of the disease.

The **prognosis** is very unfavorable. Of 39 cases collected by



Birch-Hirschfeld, only 4 recovered. Only in the early stage is recovery possible. Death supervenes from exhaustion with hemorrhages or from intercurrent pleuritis or pericarditis.

The **treatment** of a disease whose exact nature is still unknown is difficult. Good food, and hygienic surroundings are the first requisite. In the treatment of anæmia, the iodide of iron, cod-liver oil, and arsenic are the chief drugs employed. In the lymphatic form, arsenic in the form of Fowler's solution gives the best results.

### STATUS LYMPHATICUS.

*(Lymphatism; Lymphatic Constitution.)*

This condition should be differentiated from those described under the heading of Scrofulosis. Thus far it has been described only in infants and young children. Although its existence has been known for a long time, it has only lately been discussed by Paltauf, Pott, and Escherich, abroad, and by West, Rachford, and Crandall, in this country. The condition is found chiefly in children who are subject to rachitis and moderately well nourished but anæmic. They have enlarged lymph-nodes at the angle of the jaw, in the axilla, and in the groin, and are also subject to attacks of laryngismus stridulus. They have enlarged tonsils, adenoid tissue in the posterior nares, and enlargement of the adenoid tissue at the base of the tongue. On the other hand, they present none of the skin, bone, and joint-affections seen in the scrofulous or tuberculous subject. Escherich has published cases in which there were thirty attacks of laryngospasm a day. The patients also have symptoms of increased excitability of the peripheral motor nerves, such as Trousseau's phenomena and Chvostek's symptom. I have had one case in which there was an attack of laryngismus at every crying spell. The patients are in constant danger of sudden death. Several fatal cases have occurred (Escherich, Pott, Welt). Post-mortem examination in all of these cases showed that with the signs of hyperplasia of lymphatic tissue above mentioned there was enormous development of the thymus. Such were the cases of Langerhans, Paltauf, and Escherich. In the case of an infant who died suddenly in the Mount Sinai Hospital (Sara Welt), not only was the thymus enlarged to an enormous degree, but also the lymphatic tissue of the gut was the seat of great hyperplasia. The question of the cause of sudden death in these cases is a much mooted one. The old theory of pressure on the bronchi or the laryngeal nerves is now generally abandoned. Pott and Escherich have recently suggested that death is not caused by a spasm of the larynx or respiratory muscles, but by heart failure or syncope. Escherich has advanced the theory that the pathological condition of the thymus gland is the cause of a species

of auto-intoxication, which on the least provocation manifests itself in a tendency to cardiac syncope and paralysis.

The **treatment** consists in the removal of the enlarged tonsils and adenoids. In my case, as in that of Crandall, the condition of lymphatic node enlargements was vastly improved by the operation. Good food, cod-liver oil, and the preparations of iron which contain iodine, are also indicated.

### THE HEMORRHAGIC DIATHESSES.

In this class of diseases are embraced only those affections which are due to some primary change in the blood or in the circulatory apparatus. Thus conditions which are due to local disease of some organ, or the hemorrhages which follow the acute infectious diseases or drug poisoning are not included. Experimental pathology has as yet not given any clue to the etiology of the hemorrhagic diatheses. The contention of William Koch and Ajello, that they are infectious diseases or are due to some auto-intoxication, is not universally accepted. At present the clinical classification of these diatheses into the transitory forms in which are included purpura simplex, peliosis rheumatica hæmorrhagica, scorbutus, and the persistent form hereditary in character, such as hæmophilia, may be accepted. In the latter, the hemorrhage may be extensive, difficult to control, and due to some very slight cause.

#### Simple Purpura.

This is a transitory condition characterized by small hemorrhages or petechiæ, or large, irregularly shaped extravasations of blood. These are as a rule discrete, but may be confluent, and are situated in the epidermis or in the superficial layers of the cutis. Immediately after the extravasation they have a bluish-purple tinge. After a few days they become brown or greenish yellow. These extravasations are seen most frequently on the lower extremities, generally on the extensor surface. They also occur in other localities. As a rule there are few or no symptoms. There may be crops of petechiæ appearing at short intervals. In a few cases there are, after an exacerbation of the local phenomena, loss of appetite, vomiting, and general malaise. The so-called purpura cachecticorum appears on the body, abdomen, back, and upper extremities in children under two years, suffering from diarrhœa and other exhausting diseases. In the latter case there may be leucocytosis, due to the original affection. The changes in the blood in simple purpura are still to be studied.

**Etiology.**—The cause of this purpura is still unknown. It may be due to some obscure toxæmia.

The **prognosis** is very good in the primary form. In the secondary form it will depend on the nature of the original affection.





PLATE XXVIII.



Hæmophilia. Boy six years of age. Hæmatoma of the face; hemorrhage into the knee-joint. (Case of Dr. Martin Ware.)



The **treatment** will depend on the nature of the original disease. I treat the purpura itself in the same manner as cases of purpura hæmorrhagica, which will later be fully described.

### Hæmophilia.

Hæmophilia is a rare condition which may be congenital or hereditary. It becomes apparent at birth or in early infancy, and is rare in later life.

The **nature** of the affection is obscure. It is a type of hemorrhagic diathesis which is transmitted from generation to generation in the female line. It is characterized by the occurrence of uncontrollable hemorrhage after very slight injuries, and operations, and also in the absence of known traumatism.

**Etiology.**—Many theories of the cause of the affection have been advanced. They may be grouped as follows :

- (a) An abnormal delicacy and friability of the bloodvessels.
- (b) An increase of the volume of blood (Immerman).
- (c) A defect in the coagulable constituents of the blood.
- (d) Certain agencies acting as toxins on the elements of the blood, causing their dissolution (Koch).

The condition is most common in the Slavic races. Children dying of the affection show evidences of intense anæmia, but may be well nourished. Virchow has demonstrated that there is a narrowness in the arteries and also a thinness of their walls. Birch-Hirschfeld found that the endothelium of the arteries was enlarged, and that the nuclei were swollen. The blood itself shows no changes except those proper to post-hemorrhagic anæmia. The hemorrhages may occur in any region and from any organ of the body. There may be hemorrhage into joints, profuse epistaxis, intestinal hemorrhage or uncontrollable hemorrhage from the mouth or lung. The drawing of a tooth, the incision of an abscess, or a minor operation such as circumcision, may cause uncontrollable and fatal hemorrhage. In the newly born infant, there may be fatal hemorrhage from the cord. In the case pictured in Plate XXVIII., there were hemorrhages into the joints and into the face, without distinct traumatism. This case came of a family of bleeders in which there had been fatalities following circumcision.

The condition lasts weeks, months, or years—in fact, it persists during the life of the individual. Some authors believe that the female members of families thus affected should not marry.

The **treatment** is mainly prophylactic. The infant should nurse a wet-nurse, in order that the noxious influence of its own mother's milk may be lessened. Good food and fruits of all kinds should be given. All operations and traumatism should be carefully avoided.

**Purpura Hæmorrhagica.***(Morbus Maculosus Werlhofii.)*

In the prodromal period before the appearance of the hemorrhages, there may be several days of general malaise, disturbance of appetite and digestion, and febrile movement. There are anæmia, pain in the limbs, and œdema of the feet. The hemorrhages may appear without any symptoms. They are especially frequent in the lower extremities, and next most frequent in the upper extremities and on the chest, face, and trunk. They consist of extravasations of blood in the skin and subcutaneous tissue. The mucous membranes are frequently affected. Epistaxis, bleeding of the gums, bloody movements, and bloody urine result. There are ecchymoses in the conjunctiva and bleeding from the ear. The hemorrhages in the skin may be petechiæ, or irregular bluish or purple blotches which subsequently become yellowish or greenish yellow. They occur spontaneously or follow slight traumatism or pressure. There may be hemorrhages into the joints. There may be exacerbations and recurrences of hemorrhages extending over weeks. The tendency of the mucous membrane to bleed has been mentioned. The gums are spongy and bleed easily. There are hemorrhages or petechiæ on the soft and the hard palate. The hemorrhages from the kidney cause the appearance of albumin and blood in the urine. The urine is red and blood-coloring matter may be found by the turpentine-guaiac test. Hemorrhages in the brain and central nervous system may occur, causing paralysis and coma. In mild cases there is no disturbance of nutrition, but in severe ones the uræmia is marked, as is also the emaciation. The blood shows few changes. The number of red blood-cells is diminished, as is also the specific gravity. In severe cases there is a slight leucocytosis; the polynuclear leucocytes are increased, eosinophiles are few, microcytes are present, and there are a few normoblasts. The leucocytosis improves as recovery sets in.

**Course.**—The cases of ordinary severity recover. Severe cases may recover or may result fatally.

The **etiology** of this affection is still obscure. Because of its infectious nature, William Koch believes it to be allied to scorbutus and other hemorrhagic affections. His view is not supported by other writers. Ajello and Schwab regard the condition as an auto-infection or a form of toxæmia. Kolb, Tizzoni, and Babes have found bacteria in the blood of fatal cases. Others have isolated streptococci and staphylococci from the blood (Lebreton). In one of my cases there was a history of an insect-bite. The disease is rare in breast-fed infants and is more common after the age of two years than before. The infants and children attacked may have previously been in good health.



The **diagnosis** is made from the course of the affection and the size and nature of the hemorrhages. The constitutional disturbance is more marked than in simple purpura. The hemorrhages are blotches, in that respect differing from the petechiae of peliosis. The joints are not swollen, as in the latter affection.

The **treatment** consists in placing the patient in hygienic surroundings, and giving a nutritious diet with a liberal allowance of fruit and vegetable acids. In marked cases, Fowler's solution, given in moderate doses, gives good results.

### Purpura Rheumatica.

(*Peliosis Rheumatica of Schönlein.*)

Purpura rheumatica consists of an eruption of small discrete purpuric spots in the vicinity of the large joints of the extremities, and occurring especially on the lower extremities above the knee. The accompanying symptoms are pain and swelling of the joints of the lower or upper extremities.

**Symptoms.**—Slight fever is followed by the appearance of the purpuric spots and the swelling of the joints of the lower and rarely of the upper extremities. The joints are painful, as in rheumatism. At times the swelling of the joints is less apparent, but there is nevertheless tenderness on pressure. The purpuric spots are particularly numerous in the vicinity of the joints. A general urticaria may precede the appearance of the purpura. There are no heart complications. The condition of the blood is not as yet understood. There may be several crops of purpuric spots appearing at intervals of days or weeks. In other cases there are oedema of the face and enlargement of the spleen. In one of my cases there were at first slight hemorrhages from the bowel. There may be epigastric pain and tenderness in the course of the disease.

The average duration of the affection is from ten to fourteen days. There may be relapses extending over weeks.

The **etiology** is obscure. The disease occurs in children previously healthy. It is seen in older children only, and has no apparent relation to acute articular rheumatism.

The **prognosis** is good even when there are several relapses and when the affection takes a subacute course.

**Treatment.**—Rest in bed is the first requisite of treatment. A nutritious diet in which there is an abundant allowance of fruit and vegetable acids is given. Lemonade and orange-juice are especially indicated. The bowels are regulated with the salicylate of sodium given in moderate doses. A child four years of age is given grains  $v$  (0.3) three times daily. The pains in the joints are easily controlled by rest. In the subacute stage small doses of Fowler's solution are of great benefit.

### Henoch's Purpura.

Henoch in 1874 described a series of 4 cases of purpura which he classified as distinct from purpura hæmorrhagica or peliosis rheumatica. The symptoms were as follows: Children apparently in good health were attacked by a form of purpura in which there were arthritic pain, vomiting, and intense abdominal pains with bloody diarrhœa. The rheumatoid pains were accompanied by swelling of the joints. The purpura was of the hemorrhagic type—that is to say, there were extravasations of blood in the form of ecchymoses or raised exanthematic areas, not disappearing on pressure. The areas were situated on the abdomen and lower extremities. The joints affected were those of the wrist, elbow, and ankle. The intestinal symptoms consisted of repeated vomiting, tympanites, excruciating colicky pains, bloody stools, and tenesmus. One case was fatal. Such cases have been from time to time described by other observers. I have seen a case in a young infant, which ended fatally. These cases are at present regarded as due to a form of intestinal infection the exact nature of which is still obscure. They constitute a group probably belonging to the class of primary hemorrhagic affections in which is included the so-called peliosis rheumatica.

**Diagnosis of Forms of Purpura.**—It is not always possible, clinically, to assign each form of purpura to its proper class. This is especially true with young children, in whom there occur forms of purpura showing a diversity of symptoms and not fitting into any sharply defined class. Nor is it always possible at the bedside to decide whether the condition present is scorbutus or idiopathic purpura. Characteristic of both purpura and scorbutus are the hemorrhages into the skin, the internal organs, the serous cavities, and the mucous membranes. On the other hand, the frequency of hemorrhages and affections of the gums, the prodromal cachexia, the joint-affections, and the periosteal hemorrhages are peculiarly characteristic of that form of scurvy called Barlow's disease, which is seen in nurslings and young children. The purpuric affections of so-called idiopathic type, in which a purpuric exanthema is spread over the whole surface, may be called simple purpura.

In the so-called rheumatic purpura or peliosis rheumatica there is a blotchy hemorrhagic exudate over the surface in the vicinity of the joints, with pain in the joints, and gastric pains. There is always a tendency to relapses. Hemorrhages from the mucous membranes and bowels are rare, but occasionally occur.

In purpura hæmorrhagica or morbus Werlhofii there are minute or blotchy hemorrhages in the skin and internal hemorrhages from the mucous membranes, stomach, and intestines. Attempts to define sharply each of these sets of cases have been made. It is



not always possible or desirable to do so. I have seen cases of peliosis with bowel hemorrhages and gastric crises, and cases of purpura hæmorrhagica in infants, in which there were pains in the joints, evinced by the distress shown when the joints were moved. The forms of purpura regarded by Hænoch as a distinct type are classed by others as purpura rheumatica. The different classes of idiopathic purpura therefore overlap, one case frequently showing symptoms of two types. The only possible conclusion is that there may be a common cause of all forms of purpura—probably an infection.

### PERNICIOUS ANÆMIA.

This is a primary anæmia which causes progressive impoverishment of the blood and results in death. It is not common in infancy and childhood. The condition of the blood in infancy and childhood has not as yet been closely studied. The changes in the blood which have been published as characteristic of this condition in infancy and childhood are found in other states, such as the severe anæmia of rachitis and syphilis. Ehrlich is not disposed to accept these cases without question. Blood pictures which in the adult may be diagnostic of pernicious anæmia cannot be thus interpreted when found in infants and young children. Observers of note, such as Monti, Berggrün, and Baginsky, have published cases in infants and young children. I have met a case in an infant which had been bitten by a rat. After an interval, anæmia of a progressive and fatal type set in. The changes in the blood were similar to those characteristic of the same form of anæmia in the adult. Monti has collected 16 cases, 2 of which were in infants; 5 ranged from one to six years; 9 were above the age of five years. On the other hand, Ehrlich found that of 240 authentic cases, only 1 occurred in the first decade of life. That case was in a girl of eight years. In the face of such great diversity of opinion, it is wise to await the results of further research. For the purpose of reference, the following account of the changes in the blood which, according to Ehrlich, are diagnostic of pernicious anæmia in the adult, is appended:

- (a) The volume of blood is markedly diminished.
- (b) The color is at first normal, but later resembles that of beef-water.
- (c) The hæmoglobin may be as low as 10 per cent. (Fleischl). This is due to a diminution of the number of red blood-cells, for the individual cell may have a hæmoglobin content equal to the normal or above it.
- (d) There are microcytes, megalocytes, and sometimes gigantocytes. The megalocytes may constitute 70 per cent. of the red

blood-cells. They become fewer on convalescence. There are few megaloblasts, but characteristic normoblasts are found.

(e) Clumps of free granules are found in the blood. The red blood-cells may contain granules.

(f) Staining solutions produce polychromatophilic effects.

(g) The eosinophiles are normal in number.

(h) The number of white blood-cells is diminished as well as that of the polynuclear neutrophiles. The latter condition indicates serious involvement of the bone-marrow. The lymphocytes are proportionately increased.

(i) The leucocytes show no changes. Improvement is ushered in by leucocytosis.

(j) The specific gravity of the blood is diminished, as is also its coagulability.

In my case the nucleated red blood-cells were numerous.

### INFANTILE SCORBUTUS OR SCURVY (BARLOW).

(*Acute Rachitis* (MÖLLER); *Barlow's Disease, Hemorrhagic Rachitis* (FÜRST); *Scurvy Rickets* (CHEADLE); *Hemorrhagic Periostitis* (SMITH).

**History.**—Cases of this affection are described in the literature under the name *Acute Rachitis*, which was given by Möller, 1859–1862. The first definite clinical description of the disease under its present title was made by Barlow. Cheadle, Gee, and others of the English school, completed its clinical study. Northrup and Crandall have made it familiar to American physicians.

**Occurrence.**—The disease occurs chiefly in infants and in children under the age of two years. Under certain conditions it also occurs in older children and in adults. The majority of the 372 cases collected by the committee of the American Pediatric Society, occurred between the sixth and fourteenth months. The ninth month showed the greatest percentage of the cases occurring before the end of the second year. The sexes were equally affected. A second attack was recorded in a case of Holt's. In a case which I saw recently, there were two attacks.

**The Nature of the Affection.**—The nature of scurvy as it is seen in infants and children is still obscure. It is undoubtedly a form of hemorrhagic diathesis, which attacks subjects susceptible because of previous abnormal constitutional conditions and defective nutrition. There are several theories as to its exact nature. None is universally accepted. Some insist that it is a form of acute rachitis (Möller, Förster, Böhm, Steiner, Fürst, Ausset). Others contend that it is a form of scorbutus (Barlow, Northrup, Crandall, Netter, Rehn, Pott). Some of the English school regard it as a combination of scurvy and rickets (Cheadle, Gee, West). To the



latter contention Heubner, Schoedel, and Nauwerek give most support. These authors insist that the disease supervenes only in an organism already affected by slight or marked rachitis. On the other hand, there are authors who, like Schmorl and Naegeli, think that the affection is *sui generis*. Some authors have endeavored to establish a correlation with congenital syphilis. The consensus of clinical opinion, however, tends toward the acceptance of the theory of the scorbutic nature of the affection.

**Etiology.**—The essential exciting cause is not yet known. The theory of the toxæmic or infectious nature of the disease has been advocated by William Koch. Bacteria of various kinds have been found in the blood, but there is little uniformity in the results of studies. In all the cases thus far studied the nature of the diet, breast-milk, raw cows' milk, sterilized or pasteurized milk, or some artificial food, has been a strong predisposing factor. The diet has been insufficient for the nutrition of the patient, but what special element has been lacking in the food is still obscure. In the collected results of the investigations of the American Pediatric Society 10 infants were wholly breast-fed; 2 were partially breast-fed; 4 took raw milk. The greater number, 68, were brought up exclusively on sterilized milk; 16 took pasteurized milk. The others took foods of different kinds. It may be that the mode of preparing the food (raw cows' milk, pasteurized or sterilized milk) is of less importance in paving the way for the onset of this affection than its inherent composition. Cases have been cured in part by changing the composition of the food, also by substituting sterilized for pasteurized food, and *vice versa*. The very fact that breast-milk has been the exclusive article of diet in some cases should direct attention to the fact that the affection may be caused by lack of some necessary element in the diet. This view is commonly taken at present. It is interesting in this connection to consider the contention of the celebrated Arctic explorer Nansen, that if exercise and fresh air are taken, and abstinence from alcohol is maintained, scurvy on voyages will be unknown if foods are carefully sterilized and devoid of toxins and ptomains. The latter, he insists, exist in most of the milk, fish, and food eaten on voyages. Although in the most aggravated cases of scurvy that have come under my notice the diet has been sterilized milk, many infants who take that food prepared properly do not develop the disease. Some authors believe that the success of anti-scorbutic treatment with vegetable acids indicates that the organism has been for a time deprived of some essential food element. In the presence of a concrete case attention should first be directed to securing fresh food of proper composition.

**Rachitis.**—Much has been said as to the connection of rachitis with this disease. The investigations above referred to show that fully 45 per cent. of the cases occurred in infants and children who

showed clinically signs of rachitis. This does not account for cases in which rachitis may exist, but may not be apparent except on microscopic examination (Hirschsprung, Schoedel). The majority of cases examined post mortem showed the changes of rachitis (Schoedel, Schmorl).

The **morbid anatomy** has been carefully and extensively studied by Schoedel, Nauwerek, and Schmorl, whose results agree in all essentials.

The **bones** in most cases show the changes seen in rachitis. There are disturbances of growth and of bone formation. There is an increase in the width and vascularization of the cartilage zone. There are irregularity of the calcification zone, and a pathological formation of osteoid tissue. The changes at the epiphyseal junction and the periosteum are those seen in rachitis. The ribs are the bones most frequently affected, the next greatest frequency being in the bones of the lower and upper extremities. The changes caused by scurvy consist of hemorrhages into the loose vascular layer of connective tissue of the periosteum adjacent to the bone. Thus the hemorrhages are intraperiosteal and not subperiosteal, as was formerly supposed. They may be of considerable extent, either in the vicinity of the epiphyseal junction or in course of the shaft of the bone. They may form a layer several millimetres or centimetres in thickness. The outer layer of the periosteum, the fibrillar connective-tissue strata, is not the seat of hemorrhage, except in the severest cases. The layer of periosteum next the bone is thickened. The hemorrhages are both recent and old. Hemorrhages of both kinds are found in the medullary canal. The morbid changes are most marked in the ribs, next in the femur and in the bones of the upper extremities. Some of the long bones show loosening and even separation of the epiphyses and diaphyses. The infractures or fractures are of this nature. The fragments may override. In such cases the hemorrhage is great. The marrow of the bones loses its lymphoid character and becomes gelatinous.

There are subpleural and subepicardial hemorrhages. The spleen is enlarged, owing to the presence of rachitis. Slight subcutaneous hemorrhages may extend into the muscular tissue. There are hemorrhages into the mucous membrane of the hard palate and gums.

**Symptoms.**—Mild cases sometimes escape notice. An anæmic infant may cry when bathed or may favor one extremity. It may hold one thigh rigid or cry when the limb is handled in the process of diapering. Mothers at first suspect traumatism. The infant develops slight ecchymoses on the tibiæ, and is then brought to the physician. If there are teeth, there may at this stage be no swelling of the gums or of the extremities. There is no fever; there may not be any anæmia. In the severer cases the symptoms are more marked. The skin in the infant of from seven to nine months







Scorbutus in an Infant Seven Months of Age, who was Fed on Sterilized Milk. Great swelling of the right thigh and leg, due to intraperiosteal hemorrhage. Swelling of the right knee and ankle. Ecchymoses over the greater portion of the thigh, leg and dorsum of the foot. The left thigh and leg involved to a lesser degree. Complete disability of the lower extremities, especially the right, with great pain on manipulation. Swelling of the costo-chondral junction of all the ribs. Suggillation of the left eye. Complete and uncomplicated recovery.



of age acquires a pallid or greenish tinge. The infant cries when touched. One or both of the lower extremities lie as if paralyzed. If an attempt is made to move them, the infant appears to feel pain. The limb is swollen in the course of the shaft or in the vicinity of the knee or ankle, the swelling extending up the shaft (Plate XXIV.). The ribs are apparently tender. There may be one or two subcutaneous ecchymoses on the surface of the body. If there are teeth, the gums, especially those of the upper jaw, are swollen into cushion-like formations. These bleed easily and may partly conceal the teeth. If there are no teeth, the gums may appear normal, or the free border, especially of those of the upper jaw, may have a bluish, swollen appearance, which may be very slight or quite marked. The infants may have a capricious appetite, may take little of the bottle or may nurse ravenously.

The very severe cases have, as a rule, been allowed to run on for months in the belief that the infants were suffering either from rheumatism or dropsy. For some time before coming under treatment, the infant has cried when diapered or when the shoes or stockings were put on; later it becomes pale and loses ground. The appetite is poor. The thighs and the ankles begin to swell. The child does not move the extremities, which are swollen to twice or three times the original circumference. Ecchymoses appear on the surface of the swellings of the legs and thighs. Parts of the skin acquire a bluish-green, bruised appearance. Deformity occurs in the thigh, especially at the junction of the diaphysis with the head of the bones. This is due to infraction or loosening of the epiphyses at the epiphyseal line. The costochondral junction of the ribs is much swollen. There is a distinct series of very large swellings in this locality which are due to hemorrhage into the line of juncture of the rib and cartilage. Ecchymoses and sugillation appear about the orbit. The face and eyes have an oedematous, hydræmic appearance. The gums may not be at all affected, but if the infant has teeth there may be sponginess.

When the physician examines the infant, he finds that the pain produced by the procedure causes it to shriek with agony. The ribs are painful to the touch. The swellings on the thigh are uniformly fusiform, and, as a rule, hard and not fluctuating. The abdomen is tense and tympanitic. The infant has had some bleeding from the nose, but not necessarily from the bowel. In other cases there are not only hemorrhages from the bowels, but also from the kidney, in the form of hæmaturia. There may be albumin and casts in the urine.

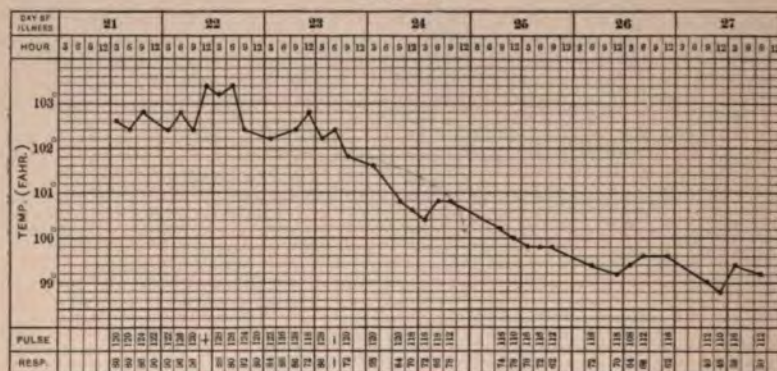
The pulse is as a rule not increased. In one case without complicating pneumonia, in which I found the respirations enormously increased, I reached the conclusion that the increase was due to the pain and extreme anæmia.

In severe cases there may be a slight temperature (Fig. 162), which may be due to resorptive fever caused by the immense extravasations of blood.

The hemorrhages in the skin may be localized in the form of minute petechiae or there may be ecchymotic blotches of considerable size. The latter may appear over the swellings along the bones.

The fractures or infractions were present in only 9 cases of the set collected by the American Pediatric Society. The gums were generally affected in infants with teeth, and were swollen and spongy in 24 cases in which there were no teeth. They may be normal in severe cases if there are no teeth, and swollen in mild ones. The symptoms in older children resemble those of adults. In one case in a child over two years of age the surgeons of a dental clinic had been consulted for an uncontrollable bleeding of the gums. The

FIG. 162.



Temperature-curve of a case of scorbutus in an infant seven months of age. Resorption fever. The chart shows the very high number of respirations as compared to the pulse. Cause of high respirations probably pain and extreme anæmia. The curve taken from the start of treatment.

child had ceased to walk on account of pains in the lower extremities, which had been interpreted as rheumatic. In older children the gums are affected, and the hemorrhages take the form of petechiae and blotches, appearing in crops over the surface of the body as in the adult. They have joint-pains and malaise.

**Prognosis.**—The disease in infants and children gives a very good prognosis if recognized and treated in time. Most cases recover. The fatal cases are those in institutions or elsewhere in which the diagnosis has not been made or in which death has been caused by some intercurrent affection, such as cerebral hemorrhage, diarrhœa, or pneumonia. In 379 cases collected by the American Pediatric Society the mortality was 8 per cent.

**Duration.**—There is no fixed duration. Much depends on an early diagnosis. Even if the disease has existed months before



a diagnosis is made, the patient may still recover. The great danger is that a hemorrhage may occur in the cerebrum or that the infant may contract an intercurrent affection through exhaustion. If allowed to continue without treatment, the disease may cause exhausting intestinal hemorrhages or hemorrhage of great extent elsewhere, with consequent anæmia and death.

The **diagnosis** of infantile scurvy presents no difficulties. The pains in the extremities, the paralytic phenomena, the swelling of the gums, the swelling in the vicinity of the joints of the limbs or along the shafts of the bones, the swellings on the ribs, and the ecchymoses in the skin and about the eye, are all characteristic. The pareses of the upper extremity are frequently mistaken for those due to syphilis. The history, and the absence of syphilitic eruptions will aid in diagnosis.

The **treatment** of infantile scurvy is simple and satisfactory. The infant is given fresh, pure milk properly modified. The milk should be given raw, and in summer should be kept well packed in ice. In addition, orange-juice and lemonade are given in the course of the day. An infant seven months old should have 2 ounces of lemonade and  $\frac{1}{2}$  ounce of orange-juice in twenty-four hours, given every two hours after each nursing. Some authors advise the giving of raw beef-juice, but it is not necessary. After two weeks the quantity of fruit juice should be reduced, but a small quantity of orange-juice should be given daily for some time. Medicines are not indicated except for the anæmia, which is best treated by doses of half a drop of Fowler's solution given three times daily, or by some easily assimilable peptonate of iron.

#### LEADING AUTHORITIES REFERRED TO IN CHAPTER X.

- "American Pediatric Society Collective Investigation of Scurvy," Trans. Amer. Ped. Soc., vol. x.  
*Barlow*: "Cases Described as Acute Rickets," Med. and Chir. Trans., Lond., 1883.  
*Bürkel*: Chronische Milzschwellung, Dissert., München, 1898.  
*Carr, W. L.*: "Case of Scorbutus," New York Med. Rec., 1892.  
*Carstensen, M.*: "Weiss-blut Körperchen beim Menschen," Jahrb. f. Kinder., 1900.  
*Cheadle*: "Cases of Scurvy and Rickets," Lancet, 1878.  
*Ehrlich and Lazarus*: "Die Anæmie," Nothnagel's Spec. Path. und Therap., Bd. viii.  
*Ehrlich, P.*: Histologie des Blutes, Berlin, 1891.  
*Escherich, T.*: "Status Lymphaticus," Berlin. klin. Woch., 1896.  
*Gee*: "Osteal and Periosteal Cachexia," St. Bart. Hosp. Rep., 1881-1889.  
*Geisler and Japha*: "Beitr. zur Anæmie," etc., Jahrb. f. Kinderheilk., 1901.  
*Gundobin*: "Morph. u. Path. des Blutes," Jahrb. f. Kind., 1893.  
*Hayem, G.*: Du Sang, Paris, 1889.  
*Hoch and Schlessinger*: "Hæmatolog. Studien," Beitr. zur Kinder., 1892.  
*Henoch, E.*: "Ueber eine Eigenthüm Form von Purpura," Verhandl. Berlin. Med. Gesell., 1874.  
*Jacobi, A.*: "Anatomy and Pathology of Thymus," Trans. Assoc. Amer. Phys., 1888.  
 — Archives of Pediatrics, 1892.

- Jaksch, V.*: "Ueber Leukämie u. Leukocytose in Kindesalter," *Wien. med. Wochen.*, 1890.
- Japha, A.*: "Leucocytose," etc., *Jahrb. f. Kinder.*, 1901.
- Koch, Wm.*: "Die Blut-Erkrankheit," *Deutsche Chir.*, Lief 12.
- Limbeck, R. R.*: *Grundriss klin. Path. des Blutes*, 1892.
- Monti and Berggrün*: *Die chronische Anämie*, etc., *Wien*, 1892.
- Morse, J. L.*: "Infantile Scorbutus," *Boston Med. and Surg. Jour.*, 1901.
- "Chronic Enlargement of the Spleen in Infancy," *Annals Gynaecol. and Ped.*, 1900.
- Northrup and Crundall*: "Infantile Scurvy," *New York Med. Jour.*, 1894.
- Northrup*: "Scorbutus," *New York Med. Rec.*, 1889; *Archiv. of Ped.*, 1892.
- Oster, Wm.*: "Sporadic Cretinism in America," *Amer. Jour. Med.*, 1895.
- "Cretinism in America," *Trans. Cong. Amer. Phys.*, 1897.
- Rotch and Ladd*: "Pernicious Anæmia," *Trans. Amer. Ped. Soc.*, 1901.
- Schiff, E.*: "Hematolog. Studien der Neugeborenen," *Jahrb. f. Kind.*, 1901.
- Schödel and Nauwerck, Möller, Barlow, Krankheit.*, *Jena*, 1900.
- Schmorl*: *Verhandl. Versammlung Deutsch. Naturforsch.*, 1899.
- "Ueber Störungen des Knochenwachstums bei Barlow'schen Krankheit."
- Stengel and White*: "The Blood in Infancy and Childhood," *Trans. Amer. Ped. Soc.*, vol. xii.
- Thomson, J.*: "Sporadic Cretinism," *British Medical Journal*, 1898.
- "Achondroplasia," *Edinburgh Medical Journal*, 1893.
- "Thyroid Feeding in Cretinism," *Edinburgh Medical Journal*, 1894.
- Triesethau, Wm.*: *Die Thymus*, *Dissert.*, *Halle*, 1893.
- Van Arsdale, W. W.*: "Cervical Adenitis," *Trans. New York. Acad. Med.*, vol. viii.
- Wentworth, A. H.*: "Association of Anæmia and Chronic Enlargement of the Spleen," *Trans. Mass. Med. Soc.*, June 11, 1901.



## CHAPTER XI.

### DISEASES OF THE BONES.

#### THE BONES.

**General Facts.**—In examining the joints, it should be borne in mind that the bones entering into the formation of the joints may be affected. The diaphysis may be diseased without accompanying involvement of the joint.

**Tuberculosis.**—In all bone lesions tuberculosis should be excluded. In infants and children, the question as to whether the existing condition is tuberculosis of the bone or syphilis is constantly arising.

Syphilis affects by predilection the long bones in the diaphysis, while tubercle affects the short bones, especially in the vicinity of the joints. In this region, also, tubercle attacks the epiphyses of the bone and may thus involve the joints secondarily.

Pain in syphilitic bone lesions is very marked, acute, and with nocturnal exacerbations; while the pain of tubercular bone lesions is obscure and indefinite, although persistent.

The swelling in syphilis is in the form of a periostitis or an ostitis involving only the bone; in tuberculosis, the surrounding tissues are affected as well as the bone, and abscess and fungous granulation result.

Syphilis rarely suppurates; the contrary is true of tuberculosis.

Syphilis of the bones does not as a rule lead to cachexia; tuberculosis of the bone eventually causes cachexia and emaciation.

There are cases in which doubt will arise as to the true nature of the bone affection. This is especially the case when the small bones of the hand are affected.

Sudden painful swelling of the long bones occurring in corresponding bones on both sides should awaken a suspicion of syphilis, even in the absence of other signs of syphilitic disease. A long bone which has been affected by syphilis will be irregularly thickened, owing to the repeated attacks of periostitis. This thickening is likely to be confounded with that caused by rachitis.

In rachitis, the bone is less painful than in syphilis and the thickening is invariably uniform and smooth. In scurvy there may be a thickening of the long bones due to hemorrhage in the periosteum. In these cases the history and also the presence of other signs of scorbutus, such as hemorrhages in the skin or bleeding of the gums, will aid diagnosis.

**Craniotabes.**—In locating patches of so-called craniotabes, the surface of the occipital and other bones of the skull is examined for deficiency of bone formation. The occipital bone will in rachitis present membranous spots more frequently than is generally supposed. The most common tumors found on the scalp are those due to traumatism at birth, such as cephalohæmatoma, tumor of the scalp with depressed bone, and tumor due to syphilis. The cephalohæmatoma is found after birth and need not be described here. If an infant falls on one side of the head from a height, a depression of the skull at once takes place. This occurs if the bones are soft and not yet completely ossified. The depression is filled with an effusion of blood and serum. A soft tumor results which may not project above the surface at all or only slightly so. Around the border of the tumor the rim of bone bordering the depression can be felt. In this respect the condition differs from the cephalic hæmatoma found after birth. In the latter, the whole tumor is raised from the surface, and on physical examination there are no evidences of depression.

**Syphilis** (see Fig. 78) may cause on the surface of the frontal and parietal bones tumors varying from the size of a hazelnut to that of a walnut. They may at first be hard and subsequently soften. They resemble abscesses, and should be differentiated from them. Tuberculosis of the bones may also cause such tumors. Tuberculosis of the skull bones in infancy is of rarer occurrence than syphilis of the skull, the cases of disease of the ear being excepted. In a concrete case, syphilis should be assumed until it can be excluded. Abscess may be diagnosed if there are abscesses elsewhere in the body. This is the case in folliculitis abscedens of Escherich. Mistakes rarely occur in these cases, since all the signs of abscess are present.

### OSTEOMYELITIS.

Osteomyelitis is an acute infectious inflammation of the structure of the bones. It is common in infancy and childhood. Of 50 cases below the thirteenth year collected by Blumenfeld, 50 per cent. were under five years of age. The sexes were equally affected.

**Etiology.**—In the majority of cases the essential cause is the *Staphylococcus pyogenes aureus*. The disease may, however, be caused by any micro-organism with pyogenic tendencies, such as the *Streptococcus pyogenes*, the pneumococcus, and the *Bacillus typhosus*. Of 90 cases collected and reported by Lannelongue, only 10 were due to the streptococcus. Lannelongue and Achard were the first to show that osteomyelitis may be caused by streptococci, in 1890. Van Arsdale and I, in 1891, published 4 cases of osteomyelitis caused by streptococci. These occurred in newly born infants or



followed scarlet fever and pneumonia. The streptococcus osteomyelitis is of especial interest to the physician, as it occurs in infants and children under two years of age. It frequently follows infection of the umbilicus in the newly born infant, the exanthemata (scarlet fever and measles), and pneumonia. It differs from the staphylococcus variety in that the inflammation of the bone is less likely to involve the medullary canal, but affects the epiphysis. There is also involvement of the joints, with suppuration. The bacteria gain access to the circulation (Garré), and to the bones through some wound such as the umbilicus, through the mucous membranes, as in ulcerations of the mouth, through some lesions of the skin such as an eczema or furuncle, or through the gut. Of the 47 cases cited above, 17 were due to trauma and 5 followed infectious diseases.

**Symptoms.**—In older children, the symptoms differ little from those of the adult subject. The femur and tibia are most commonly involved; next the humerus, superior maxilla, inferior maxilla, ileum, and radius, in the order named. In some cases the onset is sudden and the fatal issue takes place in a few days. In others, the invasion is gradual. In older children there are the regular symptoms of chill, fever, and vomiting, followed by local symptoms.

In young infants the signs of osteomyelitis are obscure. In the puerperal cases in newborn infants, the umbilicus may be inflamed for some days, after which the infant begins to cry when handled in the bath. One extremity is not moved and a joint may be swollen. Swelling of the joint may escape notice until the child is examined by the physician. After scarlet fever, the swelling of the joints is quite apparent, and also after pneumonia. In the newborn infant several joints may be swollen. In one of my cases in an infant ten months old, the elbow-joint and wrist-joint were involved, the whole radius being the seat of osteomyelitis. Similar cases have been published in this country by Gibney. The frequency of joint-involvement is a feature of osteomyelitis in children. Of 50 cases of osteomyelitis published by Blumenfeld, the joints were involved in 30. I have seen the multiple joint-suppurations most frequently in newborn infants. In all cases, there are evident swelling of the tissues about the joints and fluctuation in the joint-cavity. The joint contains pus.

The **diagnosis** is not difficult. If an infant cries when it is handled, every joint should be carefully examined. Osteomyelitis may be confounded with scorbutus. In the latter affection, the joints are painful and swollen, but do not contain fluid. In scorbutus there are ecchymoses, swelling and sponginess of the gums, and hemorrhagic lesions underneath the skin, all of which will aid in diagnosis. A history of umbilical inflammation or of scarlet fever is of great value. There are in congenital syphilis in young infants forms of inflammation about the joints which at first

simulate osteomyelitis. In such cases the infant should be examined for other evidences of congenital syphilis, such as fissures and rhagades about the mouth and anus, mucous patches, and coppery discolorations of the skin. Tuberculous inflammation in the long bones or in the heads of the bones may present some difficulties of diagnosis. A study of the case and the absence of a history of acute trouble will solve the difficulty.

The **prognosis** of acute osteomyelitis in newly born infants is bad. The majority of cases are fatal owing to the formation of multiple foci of suppuration. The prognosis is also bad in infants under one year of age. The mortality of all cases under the fifth year is 56 per cent. In older children it is 20 per cent.

The **treatment** of acute infectious osteomyelitis is surgical.

### OTITIS IN INFANCY AND CHILDHOOD.

**Frequency.**—Otitis media, catarrhal or purulent, is a very common disease of infancy and childhood. It is, as a rule, a secondary affection, but may in rare cases occur as a primary disease. Parrot first called attention to the frequency of otitis as a complication of bronchopneumonia. Netter made the first bacteriological examinations of the discharges from the ear. The subjects were 20 children whose ages ranged from nine days to two years. Kossel, Rasch, and Ponfick have investigated the frequency and nature of this affection in children. The results of their work show striking uniformity. Fully 85 per cent. of infants and children, examined post mortem, were found to have diseased ears. Most of the infants, especially in the material examined by Ponfick, had died of gastro-enteritis, acute or chronic. Some had suffered from gastro-enteritis, pneumonia, or congenital syphilis.

The **etiology** of acute catarrhal, acute suppurative otitis media and of acute suppurative mastoiditis is much the same. The nasopharynx and the Eustachian tube are normally the habitat of various forms of bacteria. This is especially the case in infants and children who have enlarged tonsils and adenoid growths. A reduction of the vitality of the individual or any acute disease favors invasion of the ear by bacilli entering through the Eustachian tube. Thus the exanthemata, especially scarlet fever and measles, furnish a large quota of cases. Diphtheria, typhoid fever, typhus fever, varicella, influenza, gastro-enteritis, tonsillitis, and simple angina, also cause a large number of cases of otitis. Pertussis, cerebrospinal meningitis, and pneumonia are complicated by this disease. Seabathing, exposure to cold, and nasal douching favor its onset.

**Bacteriology.**—The bacteria found by different observers in the otitic discharges and in the cavities of the ear include the Staphy-



lococcus pyogenes aureus, citreus, and albus, the Streptococcus pyogenes, the pneumococcus of Fränkel, the influenza bacillus and pseudo-influenza bacillus, the Bacillus fœtidus, and the Bacillus pyocyaneus (Netter, Kossel, Ponfick). The streptococci and influenza bacilli cause an especially severe inflammation, the pneumococcus a milder form. The diphtheria bacillus also causes otitis.

**Morbid Anatomy.**—In both forms of otitis and also in mastoid disease the tympanic membrane is injected and the vessels at its border are increased in size. The vessels of the hammer are injected. The epidermis of the tympanic membrane may be intact. The tympanic cavity may be filled with cellular elements. There may be a serous, mucous, purulent, or mucopurulent exudate. The mucous membrane of the tympanic cavity may be intact but injected, or may show gross defects. If the bony structures are involved, there will be necrosis of bone, especially of the tegmen tympani. There may be perforation of this structure or of the point of the mastoid process. The dura mater or sinuses of the dura may, in progressive mastoid, be inflamed. There may be cerebral abscess. If the pus does not escape by way of the Eustachian tube, it may perforate the tympanum. The exudate which fills the tympanic cavity contains epithelial cells, leucocytes, and blood-cells.

**Otitis Media Catarrhalis.**—Acute catarrhal otitis is, in a vast number of cases, simply a forerunner of otitis media purulenta or of an acute suppurative otitis. It will be convenient for the practitioner to consider these affections together.

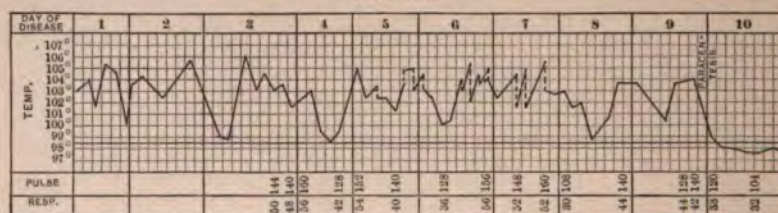
They are more common among infants and children than among adults, and may occur at the earliest period of infancy. They occur most frequently in the spring and summer.

The causation has been considered under the etiology, and is the same in both affections.

**Symptomatology.**—In young infants and in children under two years of age, the symptoms are frequently masked by those of the primary disease. In many cases, the otitis gives no special warning of its presence. Perforation of the drum and a purulent discharge are the first intimation of the condition. This is especially the case in otitis in young nurslings who have suffered from acute tonsillitis or pneumonia, but these are not the cases which the practitioner is called upon to diagnose. In another set of cases, especially in those in which otitis is coincident with gastro-intestinal disorders of a chronic type, tending to atrophy, Heermann and Ponfick have shown that during life it gives no objective symptoms although on otoscopic examination the tympanic cavity is found to be filled with pus. In cases which follow the milder types of influenza or angina, there may be a most puzzling set of symptoms which can only be referred to the ear. In these cases the physician finds, two or three days after the onset of tonsillitis or influenza, that the

temperature does not drop to the normal; it may mount to  $104^{\circ}$  F. ( $40^{\circ}$  C.) toward evening, and in the morning may drop to or within a degree of the normal. While the temperature is low the infant takes its food and plays. When it rises the infant becomes fretful, or stupid, or sleeps most of the time. There is no indication of pain. In some cases the infants perspire freely at the falling of the temperature. These simulate in many respects cases of malaria or of meningitis of the tuberculous type, except that the temperature rises higher than in the latter disease (Fig. 163). Local facial pareses may complete the resemblance to meningitis. The intermittent or recurrent curve of temperature may continue for a week or ten days. Only the careful exclusion of disease of other organs, and especially of the lungs and of the heart, will lead the physician to suspect disease of the ear. In nursing infants the bowels will be abnormal and the movements greenish, containing white curds. The temperature is, however, much higher than in any diarrhœa, and is more

FIG. 163.



Otitis media purulenta in a child eighteen months of age. Symptoms and curve simulating closely a meningitis of the basal type.

persistent and regular in its daily fluctuations. In cases of broncho-pneumonia complicated with otitis, previous to the spontaneous perforation of the drum the temperature will have shown more decided fluctuations than would occur at a late stage of the primary disease. However, in pneumonia there are few or no objective signs of the affection. Older children may have certain definite symptoms such as dull headache and pain in the ear, which, if sharp and stinging, will cause them to start in sleep, or to awake and cry out or put the hand to the ear. This last sign, so often mentioned in the text-books, I have seldom seen. There may be delirium and the fever may be quite high. Children, who can talk, complain of pain at night. There may be rushing, singing, or buzzing noises in the ear. Very characteristic is the starting of infants during sleep. Older children are out of sorts, and angry on awakening.

**Course.**—Spontaneous perforation in a number of cases occurs in a few hours or a few days after the onset of the disease. As a rule, however, pain continues with fever until artificial paracentesis of the drum is practised. After spontaneous rupture of the tympa-



num, or paracentesis, the discharge may continue, being in some cases serous or serosanguinolent, and later becoming purulent. The purulent discharge may be profuse and the disease may advance into the mastoid or labyrinth. This frequently occurs in cases of the exanthemata or in pneumonia or influenza. In severe cases, the discharge may continue and become chronic, resulting in destruction of the structures of the ear. Complications may intervene, such as facial erysipelas, meningitis, cerebral abscess, thrombosis of the cerebral sinuses, and finally in suppurative cases pyæmia may intervene. On the other hand, after spontaneous rupture or paracentesis of one or both drums, the serous or purulent discharge may gradually cease and the ears be restored without any defect of hearing. In many cases incision of the drum in the very early stages of the disease is not followed by the discharge of pus; the symptoms cease, and the patient recovers. In other cases, there is no rupture of the tympanum, although the tympanic cavity is filled with exudate, which discharges through the Eustachian tube. The pus may be swallowed and cause violent diarrhœa or pneumonia. In the cases of marasmus with otitis described by Heerman, the pus is believed to have found its way from the middle ear through the tube to the nasopharynx.

The **diagnosis** is first made from the rational symptoms. In my experience, the temperature-curve is a very useful guide in infants who give no evidence of pain. Otoscopic examination is the only positive means of making a diagnosis. There is congestion of the tympanum above Shrapnell's membrane and the long handle of the malleus. In the catarrhal cases the tympanum is red and angry or has a grayish lustre. The handle appears as a red or yellowish-white point. In some cases there are vesicles and interlamellar abscess. The exudate may cause bulging of Shrapnell's membrane or of the posterior-superior quadrant. Congestion remains long after resolution. In the suppurative cases the epithelium of the tympanic membrane may peel off. The tympanum is dull and lustreless. The auditory canal may be swollen. Perforation occurs, chiefly in the posterior-inferior quadrant. There may be pulsation of the membrane as well as bulging. The lymph-nodes beneath the ear may enlarge and that region may be very sensitive.

The **prognosis** in ordinary cases is good. In cases following the exanthemata it is grave, on account of the possibility of complications and of ultimate loss of hearing.

### THE MASTOID REGION.

**General Facts.**—The mastoid region is important on account of the frequency of mastoid disease in infancy and childhood. In

early life there is pneumatic tissue, but no mastoid cells are found. The mastoid process contains one large cell (Symington). The external wall is less thick and compact than in the adult. The petrosquamous suture is patent. The petrosquamous sinus is persistent in some cases, passes through a foramen on the inside of the skull, and appears externally behind the glenoid fossa and tympanic ring. Thus infectious material may easily be conveyed internally. In infants and children pus finds its way externally more readily through the open fissura mastoideo-squamosa.

**Etiology.**—Inflammation of the mastoid is rarely primary. The mastoid may at the outset be inflamed when there has been no antecedent otitis. As a rule, however, inflammation of the mastoid is secondary to acute or chronic otitis. The causation is identical with that of acute or chronic otitis.

Of 39 cases of mastoid disease under eight years of age, collected by Knapp, 7 occurred in the first year, and 9 in the second. The greatest frequency is therefore after the second year. It may occur as early as the second month. I have had a case in an infant three months of age. The anatomical conditions favor the occurrence of mastoid disease in infancy and childhood. The Eustachian tube is short and of large calibre; infectious material from the nasopharynx can easily gain access to the ear.

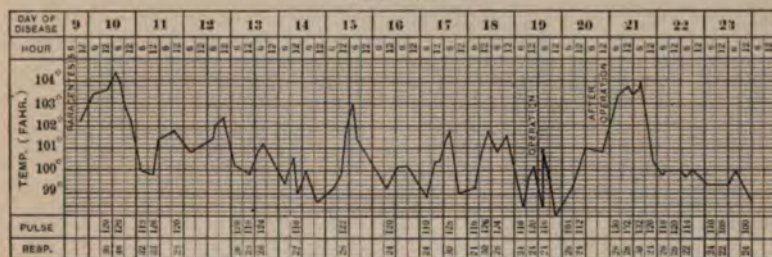
**Symptoms.**—Clinically, mastoid disease in infancy and childhood manifests itself by rational symptoms and physical signs. There may be extensive mastoid disease without any external physical signs. In one of my cases of otitis, which was observed from the outset, extensive mastoid disease in a child of three years of age did not give any external signs. The clinical symptoms are characteristic. The drum may have been perforated after otitis, or paracentesis may have been performed. After perforation, the temperature present during the preceding otitis drops to the normal. The patient is able to be up and about. The ear discharges freely. After two or three weeks there is a sudden or gradual rise of temperature, which may be slight or may reach  $103^{\circ}$  or  $105^{\circ}$  F. ( $39.4^{\circ}$  to  $40.5^{\circ}$  C.). There is restlessness at night. On inspection, the ear may not show anything abnormal. The temperature, however, continues to be remittent for several days. On otoscopic examination, there is found to be swelling of the roof of the auditory canal or of the floor of the attic. In other cases, after a very early and timely paracentesis of the drum, the patient does not do well. The child is restless at night, at intervals irritable and then playful, and starts from sleep (Fig. 164). The temperature fluctuates daily from  $100.8^{\circ}$  to  $102^{\circ}$  F. ( $38.8^{\circ}$  C.). On some days it may be normal or subnormal. The ear discharges for days, but a slight temperature continues. If the patient is an infant or a young child, it may be very difficult to ascertain whether pain is present on pressure backward over the region



of the antrum behind the ear. There is in early cases no swelling or redness behind and above the auricle. As was stated above, there may be extensive and advanced mastoid disease without external redness or swelling. In such cases the lymph-nodes behind the ear and at the angle of the jaw may be swollen and painful. Young children and infants do not complain of pain. It is only in older children that it can be noted.

Mastoid disease which follows the exanthemata, especially scarlet fever or measles, or occurs late in typhoid, shows certain characteristic clinical features. During the fifth or sixth week of scarlet fever the ears may discharge profusely. There is a daily rise of temperature in the afternoon, which is slight in some cases. The patients play in the early portion of the day, but in the afternoon appear listless, and have a slight frontal headache. As days pass, the children become stupid during the afternoon rise (sepsis).

FIG. 164.



Otitis media in a female child, three years of age. Observed from the onset. Early paracentesis, fall of temperature, then rise again. Subsequent mastoid involvement necessitating operation.

In many cases of scarlet fever, as may be seen by referring to Figs. 30 and 31, otitis is a complication. The temperature does not fall to the normal, as it should, after the fading of the eruption. There is slight aural pain at night, which is sometimes sufficiently severe to deprive the patient of sleep. In other cases the temperature drops to the normal and suddenly rises in the second week. In both these sets of cases there is an otitis which may develop into mastoid disease, or in which mastoid disease may have been present from the outset.

Körner calls attention to the fact that in late typhoid fever, chills, with rises of temperature, are, with other signs, indicative of serious mastoid disease.

**Physical Signs.**—Pain is a physical sign of mastoid disease in children. In most cases it cannot be elicited by the most skilful manipulation. In others it is impossible to come to a definite conclusion. In older children pain may be elicited by pressing the mastoid bone in a backward direction, care being taken not to press on

the auricle. The pressure should be firm and continuous. Pain in the tip of the mastoid is not of value unless there has been a perforation and phlegmon at that point (Dench).

**Otoscopic Examination.**—There is a shortening of the external canal in its posterior and upper aspect (Dench). The upper posterior wall sinks. There is bulging of the upper portion of the tympanum.

**Tumefaction** posteriorly and above the ear occurs in infants only in neglected cases. According to Dench, in these cases the pus escapes from the antrum through the aditus ad antrum into the tympanic vault. It then finds its way through the Rivinian fissure along the upper wall of the canal to the external surface of the mastoid. In children cases in which this swelling appears are less serious than adult cases. The swelling also appears much earlier in infants and children.

FIG. 165.



Mastoid disease in a child eighteen months of age. Swelling behind the ear over the mastoid. The ear is displaced away from the scalp.

**Diagnosis.**—The life of the patient often depends upon the early recognition of mastoid disease. The diagnosis in infancy and childhood should not only be made early, but should be made chiefly from the clinical symptoms of temperature, which will in its fluctuations show a septic curve. The history of the case is of service. Presence of pain is of no value in infants and young children. The daily otoscopic examination of the discharging ear will give positive evidence of mastoid disease. The signs detailed in the paragraph on symptoms are of great importance. A profuse discharge does not preclude mastoid disease. Facial paralysis is of no value. I have seen it in cases in which mastoid disease was on operation found to



be absent. Tumefaction is seen only in late cases. Redness is sometimes apparent before the appearance of swelling behind the ear.

**Course.**—If a case is neglected, pus from the mastoid may force its way through the tympanic roof and cause cerebral abscess or meningitis. It may destroy the plate (*lamina vitrea*) of the sigmoid sinus and cause thrombosis, may find its way through the tip of the mastoid along the border of the sternomastoid, and cause phlegmon, or may force itself through the *sutura mastoideo-squamosa*, causing swelling behind the auricle.

**Treatment.—Prophylaxis.**—Children can be taught to tolerate the therapeutic measures which, if catarrhal inflammation of the fauces is present, as in the exanthemata, will cleanse the parts. Thus in scarlet fever, an intelligent child will readily allow the throat to be sprayed with normal saline solution. Swabbing the throat or applying any drug locally is impracticable in children.

If the pain is excessive a mild opiate, such as paregoric, is administered. In young infants the severity of pain cannot be estimated. In older children dry heat applied externally to the ear by means of a water cushion relieves the pain. Some authors advise the application of leeches behind the ear, or the instillation of water at 110° F. (43.3° C.) into the canal with a dropper. Inflation of the ear in the early stages of otitis media has been advocated and condemned. Suction by means of a catheter introduced into the Eustachian tube is also practised. If the pain and fever are not relieved by these measures, incision of the drum is resorted to. Whether the otitis is catarrhal or purulent, it is best performed early, since damage to the ear may thus be avoided. The method of performing paracentesis of the drum is best learnt from special text-books on the subject. Ducloux advises enlargement of the opening in cases in which spontaneous rupture of the drum has taken place. Drainage by the introduction of sterilized absorbent gauze into the canal is superior to syringing. If this is not possible, syringing with 1 : 5000 bichloride is useful.

The indications for the performance of mastoid operation are protracted otitis with profuse otorrhœa, there being no tendency to resolution, acute otitis in which there is a tendency to resorption and in which paracentesis has not established drainage, also mucopurulent otitis maintained by mastoid involvement, otitis with symptoms pointing to meningeal complications, and finally otitis with complicating stenosis of the external canal, preventing drainage.

#### LEADING AUTHORITIES REFERRED TO IN CHAPTER XI.

*Babes, A.* : Bacteriol. Untersuch. über septische Prozesse im Kindesalter, Leipzig, 1889.

*Blumenfeld, F.* : Acute Osteomyelitis im Kindesalter, Dissert., Marburg, 1900.

*Cohn, F.* : "Indications for Perforating the Mastoid," N. Y. Med. Jour., 1896.

- Courmount et Jaboulay* : "Sur les microb de l'ostéomyélite," *Comp. rend hebdom. des Sciences de la Soc. Biol.*, 1890, No. 18.
- Dench* : "Mastoiditis," *Journal American Medical Association*, 1901.
- "Mastoid Complications of the Exanthemata," *Pediatrics*, 1899.
- Duel, A. B.* : "Acute Otitis Media and Mastoiditis in Scarlet Fever," *Med. Review of Reviews, Michigan*, 1901.
- Gaessler, B.* : *Mittelohres bei Scarlatina*, Thesis, Wiesbaden, 1900.
- Hermann, G.* : *Otitis Media in früheren Kindesalter*, Halle, 1898.
- Körner, Otto* : *Die Otitischen Erkrankungen*, 1896.
- Lannelongue et Achard* : "Des Osteomyélite à Streptococque," *La semaine Méd.*, 1890; *Academy of Sciences*, 1890.
- Rotch, T. M.* : "Multiple Osteomyelitis in an Infant," *Trans. Amer. Ped. Soc.*, vol. xi.
- Van Arsdale, W. W., and Koplik, H.* : "Streptococcus Osteomyelitis," *Trans. New York Academy of Medicine*, vol. viii.



## CHAPTER XII.

### DISEASES OF THE LIVER.

**Anatomical.**—The weight of the liver in infants and children is from one-twentieth to one-thirtieth of the body weight; in the adult it is one-fortieth.

**Examination.**—The liver is examined with the patient in the recumbent or semirecumbent posture. The physician may palpate for the liver or mark out the organ more accurately by percussion. In marking out the organ, the upper limit, the lower edge, and the area of superficial dulness are determined. Perfect accuracy by deep percussion is not feasible, because in order to obtain absolute dulness some force must be used, and vibratory echoes of other neighboring organs—the lungs and intestines—are thus caused. In all cases it is well to determine the upper limit of dulness at a point where the liver comes in contact with the chest-wall.

The lower border of the liver is determined by palpation and percussion. The lower border projects normally in infants and children below the border of the ribs. In the right mammillary line this projection may vary from 1 to 2.5 cm. At the xiphoid appendix the liver may project to the extent of 2 to 6 cm. and still be within the normal limits. These conditions may exist up to the tenth year. The exact age at which the liver assumes the adult dimensions has not been determined. In some adults, however, the projection below the border of the ribs is the same as in children. Since the size of the liver varies, caution should be exercised in pronouncing the organ enlarged. The gut, ascites, and tympanitic distention may obscure the lower limit of the liver both to palpation and percussion.

**Palpation.**—By palpation, the location of the lower border of the liver may be determined, and whether it is rounded or sharp, also, if the liver be enlarged, the character of the projecting portion, whether smooth or uneven. In infants and children the region of the gall-bladder is palpated, but it is difficult to determine in these subjects whether this organ is enlarged or absent. Hensch and Murchison have recorded fatal cases of increasing and persistent icterus in which there was congenital absence of the gall-bladder.

**Percussion.**—Percussion should be performed in the mid-line from the base of the xiphoid cartilage downward, in the right mammillary line from above downward, and sometimes in the mid-

axillary line. In order to determine accurately the superficial dulness, the whole extent of the dulness should be measured. This is rarely necessary except in investigations for scientific purposes. In cases of effusion into the pleural cavity, the upper limit of dulness is continuous with the dulness or flatness of the fluid. The displacement below the border of the ribs only can then be determined. In rare cases of subphrenic abscess there is an extension of the upper limit of dulness into the limits of the chest cavity, and

FIG. 166.



Method of palpating the projection of the liver below the ribs.

displacement of the lower border of the liver downward. Steffen gives the following measurements of the superficial liver dulness in the median and mammillary lines :

	Midline.	Mammillary line.
At birth . . . . .	3.5 cm.	2 cm.
At one month . . . . .	.5 "	5 "
At six months . . . . .	4.5 "	4.5 "
At one year . . . . .	4.5 "	4 "
At two years . . . . .	5.2 "	5 "
At five years . . . . .	.5 "	6.5 "
At ten years . . . . .	.5 "	6 "

These measurements also vary greatly, especially in infants under one year of age.

The following tumors and conditions simulate enlargement or disease of the liver: phantom tumor; circumscribed empyema, or pleuritic effusion; subphrenic abscess; circumscribed peritoneal effusion between the liver and diaphragm; tumors or cysts of the right kidney.

PHANTOM TUMOR is described by Murchison. It is a soft or hard epigastric tumor, which may project downward as far as the umbilicus. Whether it is dull with a tympanitic note, or tympanitic, depends on the amount of muscular contraction. There is no fluctuation or flatness. The tumor is present when the patient is stand-



ing or in the recumbent position. It disappears under anæsthesia. A tumor of this kind should not be punctured until it has been observed under anæsthesia, since there is danger of puncturing the intestine and causing peritonitis.

**EMPHYEMA.**—In simple or encapsulated empyema on the right side, the liver is displaced downward. The upper dulness extends into the pleural cavity; the lower part of the thorax may enlarge to such an extent as to press the ribs apart and cause fluctuation between them. There will be dulness or flatness in front or behind over the lower part of the pleural space, and perhaps disappearance of the respiratory murmur. It should not be forgotten that there is always a possibility of the presence of subphrenic abscess, or of abscess in the upper part or on the surface of the liver, bulging into the pleural cavity. In that case there will not only be bulging of the lower ribs, but also a continuation of dulness for a variable distance upward. The liver may be enlarged downward or not at all. If the tumor is beneath the diaphragm and displaces the liver downward, the respiratory murmur may be heard to the normal, or almost normal, limit, and yet dulness due to the upward projection of the tumor may be present.

**KIDNEY TUMOR** may extend from behind, beneath the liver, and simulate liver tumor. In such cases, the lumbar flatness extending below the border of the ribs will be a guide.

**ENLARGEMENTS OF THE LIVER.**—Enlargements of the liver in infancy and childhood present much the same physical signs as in the adult, but there are some states which are peculiar to early life.

**ANÆMIA INFANTUM PSEUDOLEUKÆMICA OF VON JAKSCH** causes great enlargement of the liver and spleen. The lower edge of the liver is rounded; the lymph-nodes are enlarged, and the blood presents certain features characteristic of this anæmia.

**SIMPLE RACHITIS** causes slight or marked enlargement of the liver, as well as real enlargement of the spleen. In some cases, the liver is not really enlarged, but may be displaced downward by the deformity of the thorax. Simple icterus usually causes enlargement of the liver, which retrogrades after a few weeks.

**CONGENITAL SYPHILIS** may cause slight enlargement of the liver which, up to the end of the second year, is present without icterus. The liver is enlarged in cirrhosis, abscess, and fatty degeneration of the organ. It is greatly enlarged in acute and chronic leukæmia.

## JAUNDICE.

(*Catarrhal Icterus; Catarrhal Jaundice; Infectious Icterus.*)

Simple jaundice is a common disease of infancy and childhood. In its simplest form, it was formerly believed to be due to an

obstruction of the common bile-duct with mucus. In recent years, the French clinicians have described a form of jaundice which they regarded as infectious. The first cases of the kind were published in 1881 by Weiss, Chauffard, and Landouzy, in France, and by Weil, in Germany. There is at present a tendency to regard all cases of jaundice in infants and children, not due to mechanical obstruction of the duct or disease of the liver, as infectious (Botkin, Hennig, Barthez, Henoch, and others). Thus simple icterus would be regarded as a mild form of infectious icterus. This view has recently been elaborated by Kissel. The theory, that errors of diet cause a catarrh of the gut, extending into the duct and thus obstructing it, finds little support. On the other hand, the theory of the infectious nature of even the mildest cases of jaundice is supported by the fact that these cases occur in groups and epidemics.

**Morbid Anatomy.**—In cases of fatal icterus, there are found atrophy and fatty degeneration of the liver cells. The interstitial tissue around the portal vein is infiltrated with small round cells. There is parenchymatous degeneration of the kidney. The whole picture resembles that of acute yellow atrophy. The mild cases of icterus have not yet been studied.

**Bacteriology.**—The bacteriology of the severer form remains to be studied. In one case Jäger found a bacillus of the proteus group in the urine.

**Occurrence.**—The disease may appear at any period of infancy and childhood. It is most common between the second and fifth years.

At present, all primary forms of jaundice may be clinically classified as follows: The very mild forms (catarrhal icterus); the severer forms; the fatal forms. It is highly probable that all are infectious in origin. The secondary forms of jaundice are not considered in this section.

**Symptoms.**—In the mildest forms there are no symptoms at the onset. In some mild cases there are vomiting and fetor of the breath, and the tongue is coated. The skin assumes a saffron hue and the conjunctivæ are distinctly yellow. The appetite is capricious; the urine is brownish and contains bile-pigment. The movements are like clay, and may have a bad odor. There is pruritus of the surface. The child may be somewhat depressed. In the very mild forms there is no febrile movement. In the majority of cases, there is rapidity of pulse and, in some cases, irregularity. In the severer forms the symptoms are more marked. The vomiting recurs at intervals, the intensity of the jaundice is much the same as in the mild forms, and the temperature may in the course of the disease be raised a degree or more. The attack may be ushered in by a chill. There is some prostration and, in a few cases, diarrhoea. The fatal cases, which were first described by Weiss and the French school,



are severer forms of infection. The symptoms of cholæmia are much more marked. There are delirium, unconsciousness, and cerebral symptoms. The pulse is greatly increased and the respirations are irregular. The patients die in an asthenic state.

The liver is enlarged in even the mildest forms. In a recent series of 20 cases of mild icterus, I found the liver enlarged from four to seven centimetres below the border of the ribs, in the mammillary line. The spleen was enlarged in most cases. The fact that in the mildest forms there is enlargement of the spleen lends support to the infectious theory of the disease. In the majority of my cases, the liver remained enlarged long after the icterus had disappeared. Kissel also found this to be the case. In some cases, three months elapsed before the liver returned to the normal limits.

**Duration.**—The disease, even in the mild form, lasts from two to three weeks. The fatal forms may run their course much more rapidly.

The **treatment** of icterus is very simple. An initial dose of calomel is given and the bowels are well evacuated. The patient is put on a milk diet, and is given a daily enema of water at a temperature of 85° F. (29.4° C.). On every second day a small dose of calomel, grain  $\frac{1}{2}$  (0.03), is given to aid the enemata. Fresh air and daily alkaline baths are beneficial. Alkaline baths are made by adding a few tablespoonfuls of sodium carbonate and an equal quantity of salt to the water.

### CIRRHOSIS OF THE LIVER.

Cirrhosis of the liver is rare in infancy and childhood. Tödten recently collected 15 cases. It has been observed to occur in early infancy (Freund, Lotze), and also in early childhood. Many cases which are undoubtedly cases of syphilis of the liver have found their way into the literature as cases of cirrhosis. The disease is more frequent in males.

**Morbid Anatomy.**—The hypertrophic is the most common form of cirrhosis in infants and children. Henoeh has published 2 cases of the atrophic variety. The morbid anatomy of the affection is the same as in the adult.

The **symptoms**, which are the same as in the adult, include enlargement of the liver and spleen, icterus, and ascites. The icterus is, as in the adult, constant.

**Etiology.**—Demme has published 2 cases in children addicted to the use of alcohol. Wilke, Gerhardt, and Murchison publish similar cases. The influence of heart disease and the infectious diseases, such as scarlet fever and measles, in causing cirrhosis of the liver is not as yet understood. Cirrhosis of the liver occurs in forms of peritoneal tuberculosis and in syphilis.

**ABSCESS OF THE LIVER.**

(*Suppurative Hepatitis.*)

This disease occurs in the newly born as a form of sepsis. Otherwise, its etiology in infancy and childhood is identical with that in the adult. It may follow a traumatism or complicate appendicitis (septic), may occur in peritonitis with pyelophlebitis, or may follow the infectious diseases. In the literature rare cases are described, in which *Ascarides lumbricoides* have caused abscess of the liver in children, by migrating into the gall-bladder through the common duct.

**Symptoms** of abscess of the liver in children are the same as in the adult.

**FATTY DEGENERATION OF THE LIVER.**

Fatty degeneration of the liver with or without enlargement of the organ occurs in forms of subacute and chronic constitutional dyscrasia. I have seen this disease in infants who died with tuberculosis, chronic or subacute intestinal diarrhoea, rachitis, Henoch's purpura, or acute leukæmia. I have also seen it in cases of phosphorus poisoning. The symptoms and signs do not differ from those seen in the adult. The diagnosis can hardly be made during life.

**SYPHILIS OF THE LIVER.**

Enlargement of the liver is common in syphilis in infants and children. The spleen may also be enlarged. There may be icterus. There may be other symptoms of syphilis, but none which can be traced to enlargement of the liver.

There are four histological forms of this variety of hepatic enlargement:

(a) The form in which gummata are found in the liver. This is rare. I have met a case in an infant sixteen months of age, in which there were gummata of the cranial and the long bones.

(b) The diffusely cirrhotic liver. In this form the connective tissue is quite evenly distributed throughout the tissue of the liver. In the lobulated liver, the connective tissue divides the organ into sections. I have seen a case in a girl eight years of age.

(c) In so-called miliary syphilis of the liver, the organ is strewn with miliary collections of round cells closely resembling miliary tubercle. The nodules are situated in the interstitial connective tissue. They rapidly undergo fatty degeneration.



**ACUTE YELLOW ATROPHY OF THE LIVER.**

This disease is extremely rare in infancy and childhood. Lanz published a case in a boy four years of age. In that there was no splenic tumor or hemorrhages, it differed from the picture in adult cases. The cases in the literature are as follows: Pollitzer, infant, one month of age; Senator, infant, eight months; Mann, infant, ten months; Greves, infant, twenty months; Widerhofer, child, one and three-fourths years; Rehn, child, two and one-half years; Löschner, child, three and one-half years; Mettenhemier, child, four years; West, child, six years; Merkel, child, six and one-half years; Rosenheim, child, ten years; Steiner, child, ten years; Folwarezny, child, fourteen years.

I have seen only one case of atrophic liver. The patient, a boy of eleven years of age, with very small kidneys, had nephritis which had appeared six years after an attack of scarlet fever. The liver dulness became gradually smaller from the time of admission to the hospital until death. At autopsy, the liver was found to have one-half the normal weight and to be the seat of marked parenchymatous degeneration.

**LEADING AUTHORITIES REFERRED TO IN CHAPTER XII.**

- Denme*: Einfluss des Alcohols, etc., Stuttgart, 1891.  
*Epstein, A.*: "Gelbsucht bei neugeborenen Kindern," Volkmann's Vortr., 180.  
*Edwards, W. A.*: "Cirrhosis of the Liver," Archives of Pediatrics, 1890.  
*Folger, C.*: "Hypertrophische Leber Cirrhose," etc., Jahrb. f. Kinder., October, 1900.  
*Jaeger*: "Weil's Disease," Zeitschr. f. Hygiene, 1892.  
*Libman, E.*: "Weil's Disease," Philadelphia Medical Journal, 1899.  
*Lanz*: "Ac. gelber Leberatrophie," Wien. klin. Wochen., 1896.  
*Neumann, H.*: "Gewöhnliche Gelbsucht," Deutsche med. Wochen., 1899.  
*Schlessinger, E.*: "Leber Carcinom," Jahrb. f. Kinderheilk., March, 1902.  
*Tödtgen, H.*: Leber Cirrhose im Kindesalter, München, 1892.  
*Weil*: Deutsches Arch. f. klin. Med., Bd. xxxix.

## CHAPTER XIII.

### DISEASES OF THE KIDNEYS.

THE weight of the kidneys is  $\frac{1}{120}$  of the body weight in the infant and  $\frac{1}{240}$  in the adult.

It is not, as a rule, possible to palpate the normal kidney in the infant or child. I have, however, seen in young infants exceptional cases in which the kidneys were situated very low down and could be easily palpated through the abdomen. I have found floating kidneys in infants and older children, but not so frequently as other observers. Comby in 1898 reported 18 cases, of ages ranging from one month to ten years of age. Steiner, Stewart, and Abt have also reported a number of cases. I believe that the displaced and fixed kidney is congenital. As the child grows and the parts are stretched, the attachments of the kidneys, congenitally low, become more relaxed. This would account for a number of cases. Jacobi believes that floating kidney in children is a congenital anomaly.

Sixteen of Comby's cases occurred in girls. A displaced, fixed kidney in infants causes no symptoms. In cases of movable kidney or floating kidney the main symptom is pain, either epigastric or radiating from the iliac region. In a girl of eight years with floating kidney, there was no difficulty in palpating the enlarged movable kidney below the liver. There were attacks of acute colicky epigastric pain, which occurred independently of the ingestion of food. The child was nervous and hysterical.

### THE URINE.

The urine, during the first ten days of life, is a limpid, clear, colorless fluid, containing sometimes epithelial cells of the bladder and urethra, and sometimes urates. If there is jaundice, it may contain biliary pigment. It has rarely a resinous odor, as in the adult.

The specific gravity during the first three days is 1010. On the tenth day it drops to 1002. It frequently happens that the newly born infant does not pass urine on the first or the second day. From the second to the tenth day it may urinate two or three times in twenty-four hours. Ruge and Robin have found that at the third month the infant urinates ten or eleven times in twenty-four hours,



passing 400 to 500 grammes in that time; at five months, 400 to 500 grammes daily; from two to three years, 500 to 600 grammes; from three to five years, 750 grammes; and from the seventh to the tenth year, 1200 grammes (Parrot and Robin).

The following tables are compiled from the results of Parrot, Robin, Camerer, and Schabanowa:

	Specific gravity.	Amount of urine in twenty-four hours.	Urea in twenty-four hours.
First day . . . . .	1010	15.0	0.10
Second " . . . . .	"	30.0	0.14
Third " . . . . .	"	60.0	0.26
Fourth " . . . . .	"	100.0	0.21
Fifth to tenth day . . . . .	1002	152-200	0.27-0.47
Thirtieth to one hundred and fiftieth day . . . . .		350	0.94
Second year . . . . .	1012	675	9.87
Third to fifth year . . . . .	1010-12	600-1200	13.9
Sixth year . . . . .	1012	1295	14.7-16.4
Tenth " . . . . .	1010.	1866	20.4

The infant passes five or six times as much urine per kilo of body-weight as the adult; the child, three or four times as much.

The infant excretes 0.3 urea per kilo of the body-weight, the child, 1.0, and the adult, 0.5. Uric acid is normally present in the urine of the newly born infant who excretes 21 milligrammes daily (Ruge.)

**Albumin** is not present in the normal urine of infants, but if the mother has during delivery suffered from eclampsia, the urine of the newly born infant may contain albumin.

**Indican** is present, in traces, in the urine of the newly born infant. It is especially constant in infants suffering from gastro-enteritis, and may be present in a number of maladies, especially in forms of suppuration. It is present in tuberculosis, but is not of diagnostic value (Zamfiresco).

## CYCLIC ALBUMINURIA.

(*Postural Albuminuria.*)

Cases of this form of albuminuria were first published by Vogel, Ultzmann, Gull, and Leube. The systematic description was first given by Pavy, by whom it has been carefully studied.

Cyclic albuminuria occurs principally in children and adolescents. The characteristic symptom is the appearance of albumin in the urine in the forenoon and afternoon, and its disappearance after a night's rest in the recumbent position. It is not present in the morning directly after rising, but appears soon after the upright position has been assumed. The quantity of albumin is not excessive. It may disappear from the urine for days and weeks, and again reappear.

The quantity of albumin does not progressively increase. The urine never contains formed elements of the kidney, such as casts.

There is no doubt as to the existence of this form of albuminuria in children, but its significance is a matter of wide diversity of opinion. Heubner has lately published some cases, and has collected from the literature 22 cases in children from one to fifteen years of age. Some authors, among them Heubner, are inclined to give a good prognosis in these cases, and to regard them as physiological forms of albuminuria. Others, among them Hensch, Leube, and Senator, are inclined to regard them as due to insidious changes in the kidney following infectious disease. It should be remembered that after influenza, scarlet fever, or diphtheria, small quantities of albumin are, at intervals, present in the urine for months and years. There may also be occasional hyaline or epithelial casts and a few blood-cells. These disappear either with or without treatment of diet and rest, but later reappear. I have seen this occur in children in good health. In one case, the child gave a history of an attack of influenza. More careful data on the subject are needed. In a given case, the urine should be carefully and frequently examined for kidney elements. It should be remembered that, in nephritis, the albumin in the urine frequently takes a cyclic course (Senator).

#### **ŒDEMA OR HYDRÆMIA WITHOUT KIDNEY LESION.**

Weak infants who have suffered from chronic gastro-enteric catarrh have swelling or an œdematous condition of the dorsum of the feet and ankles. There may be slight anasarca elsewhere. There is no real kidney lesion; the condition is one of hydræmia. The changed state of the tissues, including the vessels and blood, allows of a transudation of serum into the subcutaneous structures. On examination, the urine is found to be abundant and of low specific gravity, but without evidences of nephritic degeneration. In children of two years of age this condition of slight subcutaneous œdema occurs in simple anæmia of a severe type. In these cases the skin is yellowish, the ears have a waxy clearness, the eyes have an œdematous appearance, and the lips, hands, and feet are puffy. The condition is known as hydræmia or hydræmic anæmia.

#### **DYSURIA.**

Dysuria, or difficult and painful micturition, is a condition in which there is partial obstruction to the free flow of urine from the urethra. It is not uncommon in young infants and children, and may be due to a variety of causes. If lithiasis is the cause, there is not only pain in passing the urine, but there may, in the intervals, be acute attacks of



pain, due to the passage of calculi along the ureter. Examination of the urethra in the male often results in finding a calculus of very small size in the anterior penile urethra. In lithiasis, there is sometimes very painful micturition without the formation of calculi of any size. The minute crystals of uric acid cause a smarting sensation as the urine passes over the urethra. In febrile states with concentrated urine, the acidity of the urine, and the excess of uric acid with free crystals, cause painful micturition.

Simple or gonorrhœal inflammation of the urethra may cause difficult and painful micturition. Dysuria is painful at the onset of vulvovaginitis. Another condition of congenital origin, which was described by Bokai as **cellular atresia of the labia**, is a very common cause of dysuria. It is seen in very young female infants. From birth, the urine is passed in drops and with great straining and pain. In some cases it is passed without pain, but the condition of atresia attracts attention. On gently separating the labia majora a thin pinkish-white membrane is seen to occlude the introitus vaginae completely. At the urethral end of this membrane, a very minute opening is seen, through which the urine filters. These membranes can be divided by means of a dull director. It is then seen that the hymen and urethra are directly behind the membrane. The operation of dividing the membrane is exceedingly simple, and causes little or no bleeding. Bokai has described a similar condition in boys, which is somewhat less common. It is a cellular adhesion of the prepuce and glans penis which not only causes false phimosis, but also difficult and painful urination. He found that in the newly born infant the prepuce was sometimes adherent to the tip of the glans penis, and that across the opening of the meatus there was a very thin membrane. In other cases, this membrane was ruptured, but the prepuce still remained adherent to the glans in front, while behind at the corona glandis there was retention of smegma and consequent painful inflammation.

The **treatment** is division and separation of the cellular adhesions. Other abnormalities in infant boys, among them diverticula of the urethra, may cause dysuria.

## HÆMUTURIA.

Hæmaturia is the passage of blood and its elements into the urine, in which blood-cells and coloring-matter are found. The condition may occur in the following states :

(a) Acute nephritis of all forms, especially those complicating the infectious diseases, such as scarlet fever, measles, typhoid fever, and malarial fever.

(b) Calculi, renal or vesical.

- (c) Malignant growths of the kidney—sarcoma and carcinoma.
- (d) Growths of the bladder—polypus.
- (e) Traumatism in the region of the kidney.
- (f) The ingestion of drugs.

The color of the urine varies from a slightly smoky amber to a deep brownish-red. There may be a deposit of blood-cells and clots in the urine. Pure blood with clots is seen in cases of malignant tumor of the kidney and calculi of the kidney or bladder. Smoky urine is seen in cases of nephritis and drug-poisoning.

### HÆMOGLOBINURIA.

Hæmoglobinuria is a condition in which the urine contains the coloring-matter of the blood, but, except in rare cases, no red blood-cells. The urine is reddish or brownish, and has a high specific gravity. It contains albumin. By spectral analysis, the spectrum of the blood coloring-matter is obtained. According to Hoppe-Seyler, methæmoglobin and not hæmoglobin is often the coloring-matter present. There are few blood-cells and no detritus. Several theories have been advanced to explain the appearance of hæmoglobin in the urine, that of Ponfick being generally accepted. According to that author, either the blood-cells are destroyed by some vicious agent or ferment (Ehrlich) and the hæmoglobin is thus let loose into the circulation, or the hæmoglobin is dissolved out of the blood-cells and passes into the circulating plasma, leaving the cells behind as so-called "shadows." Whatever the real cause, the exciting influences are as follows:

(a) Cold or exposure to wet. Hoff and Demme have published cases of children with paroxysmal hæmoglobinuria following such exposure.

(b) Drugs, such as arsenic, phosphorus, potassium chlorate.

(c) The infectious diseases, such as malaria and scarlet fever, erysipelas.

(d) Hæmoglobinuria has been observed in cases of burns.

(e) Baginsky has observed hæmoglobinuria in children with nematodes.

In the paroxysmal form, each attack is preceded by a chill and followed by dyspnoea, palpitations, cyanosis, and severe symptoms of collapse. The attack may last a few hours or a few days, the duration depending on the course of the primary affection. This form has been especially observed to occur in pernicious malaria.

The **treatment** consists in the management of the primary exciting conditions.



**RENAL CALCULI.**

(*Uric Acid Infarction; Lithaemia.*)

So-called uric acid infarction is found in the kidneys of over one-half the infants who die in the first weeks after birth. These infarctions are seen in the medullary portion of the kidney as golden-yellow or brownish rays which are broader toward the papilla. Ebstein found isolated deposits in the cortex. The infarctions consist of uric acid (Schlossberger). They are supposed to be due to the destruction of tissue rich in nuclein (cells) (Kossel and Horbaczewski). They are found in weaklings, and more often in infants who have been born living than in stillborn infants. During the first weeks of life they are washed out by the urinary secretion. Hence the increased uric acid excretion at that time. As a rule the condition gives no symptoms. It is not uncommon for the diapers of the infants to be stained red, and in older children there may be the so-called brick-dust deposit in the urine. In these cases there may be a history of severe colicky attacks. In other cases the infant or child experiences pain on urination and cries piteously. Some older children will run about in pain and grasp the penis. In all such cases I examine the diapers for concretions. Failing to find these, I carefully examine the urethra. In several cases I have found an oval calculus of the size of a rice-seed, imbedded in the canal of the penile portion of the urethra. These cases have attacks of pain extending over months, and probably caused by the passage of the calculi from the kidney through the ureter, the bladder, and urethra. The calculi are easily extracted with long-bladed forceps. In one of my cases of hæmaturia, in a boy three years of age, there were several attacks lasting for days, but no distinct history of pain. The urine contained blood coloring-matter, some blood-cells, and a few hyaline casts, which it was difficult to find. The diagnosis was obscure until a few small calculi were found in the urine. Urotropin given in small doses caused a cessation of symptoms.

**ACUTE NEPHRITIS.**

- A. **Acute Parenchymatous Nephritis or Acute Exudative Nephritis** (DELAFIELD); **Tubular or Glomerular Nephritis.**
- B. **Acute Diffuse Nephritis or the Acute Productive Nephritis** (DELAFIELD).

The **etiology** of both forms of acute nephritis is the same.

There is scarcely an acute infectious febrile disease which may not give rise to acute nephritis. It complicates or follows scarlet

fever, measles, influenza, diphtheria, infectious angina, pneumonia, rheumatism, typhoid fever, sepsis of all kinds, variola, parotitis, malaria, and congenital syphilis. The frequency in scarlet fever of the oedematous forms with anasarca has led to the belief that this disease was most often complicated by nephritis. If the parenchymatous form is included, the condition will be found to be very frequent in other infectious diseases, but it is often unrecognized. The essential causes of acute nephritis are micro-organisms or their toxins. Thus in the various diseases, the *Diplococcus pneumoniae*, the typhoid bacillus, streptococci of various kinds, staphylococci, and the *Bacillus pyocyaneus*, have among other bacteria been found in the kidney. On the other hand, in diseases such as diphtheria, the toxins of the bacteria are the cause of the parenchymatous or diffuse nephritis (Fürbringer, Roux, Councilman). If the toxins are formed in the body, the infections are said to be autochthon or endogenous. The irritating toxin may be introduced from without, as in chloroform or ether narcosis, and the ingestion of drugs (ectogenous). The rôle played by cold as a causative factor is still a matter of speculation. Its mode of action, whether reflex, through the circulation, or by causing changes in the blood, is still obscure.

**Morbid Anatomy.—Acute Parenchymatous or Exudative Nephritis** (Delafield).—This is an acute inflammation of the kidney, in which the principal changes occur in the epithelium of the tubules and Malpighian tufts. The kidneys are larger than is normal, and succulent. The capsule can be stripped from the surface, which is red, grayish, and punctate in spots. All the changes are most marked in the cortex of the kidney. Evidences of inflammation are found in the tubes, stroma, and glomeruli. The epithelium of the tubes is flattened, granular, and fatty, or in a condition of coagulation-necrosis. The lumen of the tubules may be empty or may be filled with desquamated epithelium or with coagulated masses (casts) of a hyaline character. Delafield describes the tubes, in severe cases, as filled with leucocytes and blood-cells. The tubes may be uniformly dilated.

The changes in the glomeruli may be so slight as to be scarcely noticeable. The cavities of the capsules sometimes contain coagulated matter and red and white blood-cells (Delafield). In marked cases, there are desquamation of capsular epithelium and increase of nuclei. The swelling and proliferation of cells sometimes change the appearance of the tuft so that the outlines of the individual capillaries are lost. The stroma is infiltrated with serum, and in severe cases there are in the cortex small collections of white blood-cells (pus).

The changes in *acute diffuse nephritis*, or the acute productive nephritis of Delafield, are more serious and permanent. According to Delafield, the kidneys are large, and at first smooth and later rough. The cortex may be mottled yellow and red; the pyramids



are red. In this form of nephritis there are the changes found in exudative nephritis, and also a growth of connective tissue in the stroma and an increase of the capsule cells of the Malpighian bodies. These changes involve symmetrical strips in the cortex, which follow the lines of the arteries (Delafield). The Malpighian bodies show an enormous growth of capsule cells with compression of the tufts. If the nephritis is acute, the interstitial tissue is augmented with newly formed cells and basement substance. There is a new growth of connective tissue between the tubules; the walls of the arteries are thickened. In the capsule of the Malpighian tuft, there is a growth of cells which compress the tuft of vessels. These and the vessels are in turn converted into small balls of fibrous tissue (Delafield). In addition there may, in the acute forms of nephritis, be hemorrhages throughout the kidney substance.

**Symptoms.**—In the forms of parenchymatous nephritis which complicate the febrile infectious diseases, influenza, pertussis, angina, and gastro-enteritis, either the symptoms of the primary disease mask those due to the kidney lesion or the nephritis may be so mild as to give no symptoms. Thus in the parenchymatous nephritis which complicates or follows influenza, there are after the attack has passed no symptoms referable to the kidneys, yet on examination the urine shows a trace of albumin, hyaline and a few epithelial casts, and an occasional red blood-cell. In these cases there is no oedema of the tissues, no headache, and the children are apparently well except for the changes in the urine. These may at first be quite marked. After a few months the albumin may only appear occasionally; the casts and blood disappear for weeks and then reappear. For weeks or months the children may have no constitutional symptoms. In the parenchymatous nephritis, which is seen in severe forms of gastro-enteritis and dysentery, the signs in the urine of marked nephritis are albumin, casts of all kinds, and blood-cells (Parrot, Fischl, Czerny, Koplik, and Morse). Although Czerny traces a certain form of dyspnoea to the influence of uræmia in these cases, no distinct set of symptoms due to the kidney can yet be formulated. It is true that there are terminal anasarca, suppression of urine, and vomiting, but the presence of all these may be explained by the severity of the intestinal lesions.

**Changes in the Urine.**—In all the diseases above mentioned, the parenchymatous nephritis may in infants and children be evinced by diminution of the quantity of urine, or the presence of a trace of albumin, or a few hyaline or epithelial casts and blood-cells. The quantity of urine may, however, be normal. In other cases, the albumin is more marked and the casts much more numerous. Renal epithelium is also present. Leucocytes are rare.

In the diffuse or productive form of nephritis in infants and children, the symptoms are marked. In some forms of nephritis

complicating scarlet fever the lesion never advances beyond the parenchymatous stage, and at that period the symptoms are either not present or not noticeable. If the nephritis is more marked, however, it is noticed at the end of the third week that the patient is somewhat pale, that the face is a little swollen especially about the eyes, and that there is very slight œdema of the general surface. In these cases it is possible at the end of the period of eruption, to find a slight trace of albumin in the urine and a few hyaline and epithelial casts. With the onset of the anasarca the albumin increases in quantity, the casts in number, and a few blood-cells are found. The quantity of urine is diminished, but in the mild forms not markedly so. A boy of six years may pass half the normal quantity. There is no headache, and only a few obscure pains in the joints. There is occasionally slight pain in the region of the kidney. The temperature is normal or may at intervals of several days rise a degree or a degree and half above the normal. The nephritis is probably of the mild diffuse type. In three weeks the mild anasarca disappears, the anæmia improves, and the urine becomes normal.

In the more severe cases there is a rise of one or two degrees in temperature, and the patients have marked general anasarca. If old enough, they complain of headache, they vomit, and show marked decrease in the number of respirations and pulse, the irregularity of pulse being of a purely uræmic character. In some cases there are effusion into the chest (hydrothorax) and abdominal ascites. The quantity of urine is much diminished, there being only one or two ounces in twenty-four hours. The specific gravity is high; the urine contains blood, leucocytes, and casts (hyaline, granular, and epithelial), with blood cells. Under treatment, the vomiting, headache, and anasarca subside, the quantity of urine increases, the number of casts and blood-cells diminishes, and the patient makes a good recovery. In other cases the initial anasarca becomes more marked, there being considerable œdema of the whole surface; the urine is entirely suppressed; the vomiting and headache increase; convulsions set in; there are several attacks of eclampsia; the patient becomes comatose, and may die of uræmia, or after one or two attacks of eclampsia, the symptoms may abate and recovery take place.

There is a very fatal form of diffuse nephritis which occurs on the fourth or fifth day of malignant scarlet fever. On the third day, at the height of the eruption, the patient passes into a delirious, semi-conscious state. The quantity of urine is much diminished; its specific gravity is high; casts of all kinds and blood are present. The urine may finally be totally suppressed. There is no œdema of the surface. Coma and convulsions set in. The patient succumbs to the intense general toxæmia and to its effect on the kidneys. In



these cases the kidney symptoms cannot be separated from those caused by the general intoxication.

**Individual Symptoms.**—**THE VOMITING.**—The vomiting in scarlatinal nephritis is rarely distressing, and subsides in a short time. It is not a constant symptom, nor is it of serious import.

The **HEADACHE** is not a very marked symptom in children.

**CEDEMA** is present in a large proportion of cases, and is marked in the severe ones. It may occur with hydrothorax, ascites, and hydropericardium. It may affect only the face, or the lower extremities alone. It may be so intense as to cause bursting of the skin and the escape of serum through the fissures. It may affect one half the body more than the other (Henoch). Under all these conditions, the outlook is serious.

The **PULSE** is sometimes inordinately slow. It may be more rapid than normal, and may show marked irregularity.

The **HEART** may, as was pointed out by Henoch and Friedländer, be the seat of hypertrophy and dilatation. There may be complicating endopericarditis.

The **LUNGS** may be the seat of pneumonia, or œdema of the lungs may suddenly develop. There may be complicating pleuritis.

There may be **CONSTIPATION** or more or less diarrhœa.

There are cases in which the **TEMPERATURE** is normal or sub-normal during the whole course of the disease. In the cases in which there are sudden eclamptic seizures, the temperature may mount to 104° F. (40° C.) during the attacks. On account of the rupture of a bloodvessel in the brain during the eclamptic seizures, there is in many cases, after the subsidence of the uræmic symptoms, aphasia, or hemiplegia of a more or less permanent nature.

Patients with nephritis succeeding scarlet fever develop fainting spells with cyanosis, galloprhythm, and all degrees of cardiac weakness. It is difficult in such cases to know whether to attribute these symptoms to the nephritis or to myocarditis which is the result of the scarlet fever.

**THE URINE.**—The general characteristic features of the urine in acute diffuse nephritis of scarlet fever have been given. Suppression may take place suddenly. The urine may not have contained coagulable albumin or casts, and the quantity may have been normal. The common notion that uræmia or eclampsia can supervene only if the quantity of urine is diminished, is erroneous. Even if the quantity is above the normal and the urine contains little albumin and few casts, eclampsia may supervene with fatal results. An increase in the quantity of urine above that of the normal is an unfavorable symptom. The quantity of urea passed is always the crucial test. There are cases in which blood appears in the urine and in which there is true hæmoglobinuria, which may give rise to irritation of the kidney. In other words, the hæmoglobinuria is

primary, the nephritis secondary. The quantity of albumin in the urine varies greatly; it may only amount to a trace or be sufficient to cause the urine to become solid when boiled.

### The Primary Forms of Acute Nephritis.

The question has arisen: Can nephritis be primary? If nephritis is the result of some form of infection, it cannot be primary. Henoch, Heubner, Bouchut, Bartels, Loos, and Holt have published cases in nurslings, the origin of which could not be traced. These occurred in infants from five weeks to one and a half years of age, who suddenly developed marked anasarca and vomiting, with suppression of urine. Some of the cases had a febrile movement of a remittent type. The majority of them were fatal. Their exact nature is still unknown. Uhlenbrock has recently collated all the cases in the literature, but has thrown no light on the subject. On autopsy, a few cases have shown a parenchymatous nephritis.

**Course.**—The majority of cases of parenchymatous or exudative nephritis recover. The prognosis of the diffuse or productive form is more serious, but in exceptionally mild cases recovery may take place. Other cases make an apparent recovery. After the symptoms of œdema and anasarca have disappeared, anæmia remains. The albumin in the urine may disappear and reappear. In six months or a year, general anasarca may set in with all the symptoms of an acute exacerbation of the disease. The patient may eventually recover from the attack, but as a rule others of the same kind follow, and the condition of chronic nephritis results.

**Duration.**—The acute forms of parenchymatous or diffuse nephritis last from two to six weeks. The parenchymatous forms are sometimes evanescent, the marked symptoms lasting only a week.

### Chronic Diffuse Nephritis.

(a) *Chronic Productive Nephritis*, (b) *Chronic Nephritis without Exudation* (DELAFIELD).

The forms of chronic diffuse nephritis are the same in childhood as in adult life. They usually occur late in childhood. Thus one case of chronic diffuse nephritis in a girl of fourteen years of age dated from an attack of scarlet fever at the age of eight years. At autopsy there was found a diffuse nephritis of the productive variety (large white kidney). In another case, a boy of twelve years, with diffuse nephritis of the non-productive variety (small cirrhotic kidney), had had an attack of scarlet fever at the age of five years. He had no anasarca in the course of the nephritis. Active symptoms of headache and vomiting appeared a year and a half before his death. The quantity of urine was above the normal and there were a few hyaline casts. At autopsy a small kidney was



found. Thus there may in children be two forms of chronic nephritis following scarlet fever or any other infectious disease. Adults present symptoms referable to the eye, such as neuroretinitis, which I have not met with in children, and which must be exceedingly rare. Neither have I seen in children the emphysema met in adults. The heart may be hypertrophied and dilated in children as in the adult. They may have endocarditis and pericarditis with pleurisy.

**Treatment.**—The forms of parenchymatous or exudative nephritis which so frequently occur as accompaniments of the acute febrile disorders, pneumonia, typhoid fever, influenza, etc., need little or no treatment. There are no symptoms referable to the kidney. Nephritis accompanying acute gastro-enteritis is best treated by remedies directed toward the primary affection. The quantity of urine is sometimes diminished. It contains casts of all kinds. Rectal enemata of saline solution at a temperature of 108° F. (42.2° C.) are then of great utility, not only in supplying fluid to a depleted circulation, but also in stimulating the circulation and therefore the kidney secretion. Drugs which might still further compromise the condition of the kidney should not be given for the intestinal affection. Hot baths are of great utility, 105° F. (40.5° C.).

In the partial or complete suppression of urine seen in the first few days of the malignant forms of scarlet fever, more active treatment is required. When the temperature is high, the pulse rapid and weak, the patient unconscious or delirious, and the urine diminished or suppressed, I administer high and large rectal enemata of water at a temperature of 108° to 110° F. (42.2° to 43.3° C.), as recommended by Kemp. These should not be given to children with a double-current tube, but simply as enemata. About a quart of saline solution is thrown into the rectum at very low pressure. A fountain bag syringe is utilized for this purpose. These enemata stimulate the heart and circulation and supply the system with normal fluid. To stimulate the skin, the warm baths are preferable to cold ones. Patients are frequently much depressed by cold packs or baths given to reduce the temperature. The temperature of the bath should be at least 105° F. (40.5° C.), and the patient allowed to remain in it five or ten minutes, according to the state of the pulse.

In acute cases the anasarca will, as a rule, take care of itself. If it is extreme, Senator advises the administration of diuretics in acute as well as chronic nephritis. Some authors recommend diuretin and digitalis in form of infusion, a drachm being combined with an agreeable alkali, such as citrate of potassium. The pulse should be watched. If it is low, the digitalis is suspended. I do not utilize whiskey or alcohol in these cases. In acute diffuse nephritis and

in productive nephritis similar to that of scarlet fever, the uræmic symptoms, the œdema, and the kidneys are treated. Vomiting is a uræmic symptom which is prominent at first. If the patient vomits everything ingested, no food should be given by mouth. The patient is nourished by rectum by means of somatose or nutritive enemata. The headache needs little treatment. Bromide and a small dose of chloral or trional are given for restlessness at night. In the forms of nephritis, generally subacute, in which there are œdema amounting to anasarca, and diminution of urine, baths and diuretics are beneficial. The anasarca is sometimes scarcely noticeable, and the quantity of urine little diminished. There are usually a few hyaline and epithelial casts, and also blood-casts. The patient is kept in bed and put on a milk diet. The bowels are kept open by means of Vichy water given in liberal quantities, or by Carlsbad salts. A child between four and six years of age should take half a drachm of the salts once a day. Some mild diuretic, such as citrate or acetate of potassium, is given. The pulse may be 80 or 90, and digitalis is therefore not given. Under this mild therapy the anasarca subsides, the albumin diminishes, and the urea and quantity of urine increase. Milk also tends to increase the quantity of urine. A bath at 104°–105° F. (40° C) is given every day or every second day according to the indications. The diaphoretic effects of vapor baths are less marked. In some of the severer cases the urine is greatly diminished, the anasarca extreme, the pulse and respirations are increased, and the temperature may be elevated. The anasarca is then treated by a daily warm bath, in which the patient remains for five minutes, and is then wrapped in a warm dry blanket to promote diaphoresis. A warm rectal enema at the temperature above mentioned is given twice daily. The kidneys are stimulated by means of digitalis and acetate, citrate, or tartrate of potassium. The digitalis is given in form of the infusion, ʒss–ʒj with 3 to 8 grains of the potassium salt, three or four times daily. The pulse is closely watched and not allowed to fall too low. The bowels are kept open by the daily administration of cathartics. If, as frequently happens, the heart becomes weak, sparteine or liq. ammoniæ acetatis and nitroglycerin may also be given. I do not administer preparations of musk or camphor in nephritis. Convulsions are best controlled by means of chloroform. Warm baths and high warm enemata are also useful. Bromide and chloral are also given by rectum, as in ordinary eclampsia.

In convalescence the question arises, When shall diuretics be discontinued? As soon as the quantity of urine is above the normal, they are of no further value. The baths and enemata are continued as long as there is the least œdema of the surface. Warm enemata should not be continued after the urine has increased to the normal amount. Ordinary enemata are then given for the purpose of aiding



the cathartics in keeping the bowels open and clear of fecal accumulations.

Rest in bed should be continued until there is no palpable albumin reaction. Meat and vegetables are then added to the diet list. If anæmia is present, a readily assimilable form of iron, such as the peptonate, is given. Casts will appear in the urine far into convalescence. The patients may, however, be allowed to be up if they bear the change well. A too protracted stay in bed is sometimes exhausting in summer. If symptoms of anasarca and other signs of nephritis recur, the treatment is the same as in primary acute attacks. The treatment of chronic nephritis in children does not differ from that followed in the adult.

### NEW GROWTHS OF THE KIDNEY.

Thirty-eight per cent. of all the reported cases of kidney tumors occurred in children (Döderlein, Lewi). The following growths are here considered: 1. Cysts of the kidney; 2. Tuberculosis of the kidney; 3. Carcinoma of the kidney; 4. Sarcoma of the kidney.

#### Cysts of the Kidney.

Cysts of the kidney are in children usually of congenital origin. They are formed in the second half of intra-uterine life. They are bilateral, only 1 in 60 being unilateral (Léjars). The kidney is made up of greater and smaller cysts. The cystic formations may be present to the entire exclusion of kidney tissue. The cysts may attain the size of a child's head and seriously obstruct delivery. They are of anatomical interest only, since infants with such cysts present other abnormalities and die soon after birth.

#### Hydronephrosis.

Hydronephrosis is either congenital or acquired. If acquired, it occurs late in childhood. The congenital form is due to stenosis in some part of the urinary tract. Hydronephrosis is as a rule unilateral. If it occurs after birth, it may be due to obstruction by calculi or to uric acid infarction of the kidney. The healthy kidney is physiologically enlarged. The acquired form is due to obstruction by calculi or to tumors pressing on the ureters. At first the pelvis of the kidney, then its tissue is encroached upon in the gradual dilatation. Finally the shape of the kidney is lost. There is a large fluctuating tumor which may or may not be painful. When it is punctured, there is withdrawn a fluid which contains albumin, epithelium, urea, uric acid, and which has a low specific

gravity. In some cases there occurs what is known as intermittent hydronephrosis. The contents of the tumor are emptied spontaneously, but reaccumulate. The diagnosis rests on the presence of a fluctuating tumor containing the fluid, and urine constituents. Cystoscopy may in some cases reveal obstruction of the ureters.

Cysts must anatomically be differentiated from the condition of hydronephrosis. Cysts are new growths (Senator); in that respect they differ from the cystic condition of hydronephrosis. It is not possible clinically to differentiate congenital cysts of the kidney from congenital hydronephrosis.

### **Sarcoma of the Kidney.**

Sarcoma of the kidney occurs in children as a primary growth. In the statistics of Rosenstein and Senator two-thirds of the cases occur before the tenth year. It is more frequent in females. The left kidney is more commonly affected. Sarcoma occurs in the newly born infant. The presence of muscle, bone, and cartilage tissue in these growths supports the theory of their congenital origin (Jacobi). The anatomical nature of the growth varies widely. It may be round-celled or spindle-celled sarcoma, a fibro-sarcoma, myo-sarcoma, angio-sarcoma, melanotic sarcoma, or adeno-sarcoma. There may be metastases. The tumors sometimes attain a weight of fifteen pounds.

The **symptoms** do not differ materially from those of carcinoma of the kidney, nor is sarcoma of slower growth. In many cases the pain, hæmaturia, and tumor follow a traumatism. Hæmaturia is not, as in carcinoma of the kidney, a constant symptom. I have seen cases of both carcinoma and sarcoma of the kidney in young children without hæmaturia or growth elements in the urine. Ascites is present in more than one-half the cases (Lewi).

**Diagnosis.**—A malignant growth in a child may be surmised to be a sarcoma, since those growths are more frequent in children than carcinomata. Swelling of the lymph-nodes may be present in sarcoma as well as in carcinoma. Histological elements in the urine are rare. Von Jaksch has mentioned the presence of small round cells (sarcoma cells), but their significance is not as yet determined. Puncture for diagnostic purposes is dangerous, and if performed at all should be done posteriorly in the lumbar region (extraperitoneal). In sarcoma of the kidney, as in all growths of that organ, the colon is pushed in front of the growth (Plate XXX.).

### **Carcinoma of the Kidney.**

Of 449 cases of carcinoma of the kidney (Rohrer, Ebstein, Lachman), 157, or almost 35 per cent., occurred in children under the tenth year. Monti tabulated 50 cases, and found that more than



PLATE XXX.



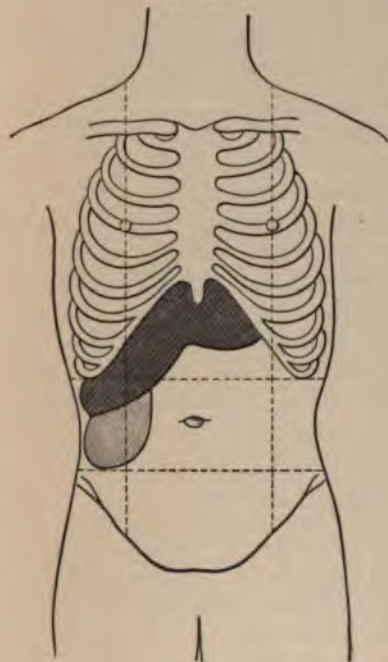
Sarcoma of the Kidney. Child six years of age. Irregular contour of the abdominal tumor.





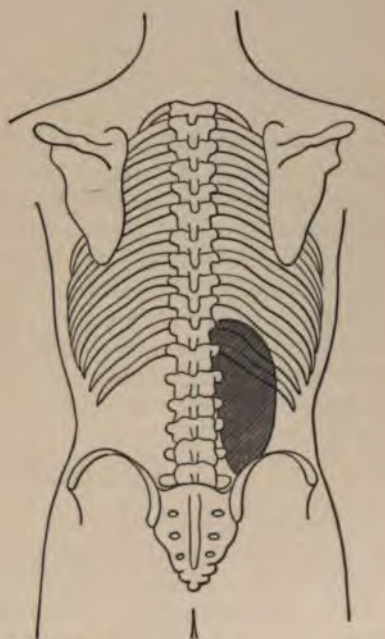
50 per cent. occurred in children under the age of two years. The youngest patient was twelve months of age. It is more frequent in males. As a rule the right kidney is affected. In children, the growth is apt to attain great size. Guillet found that the average weight was eight and one-half pounds. By reason of the great weight of the growth, the kidney may sink from its normal position and lie transversely across the vertebral column. The growth is a primary one. The medullary carcinoma is the prevailing type; the scirrhus is next in order of frequency. The disease may be secondary to carcinoma of the suprarenal capsule or of the retroperitoneal glands. The liver, the lungs, and the inguinal lymph-nodes may be secondarily involved.

FIG. 167.



Anterior palpable tumor beneath the liver.

FIG. 168.



Posterior area of flatness in the lumbar region, giving a palpable tumor between the border of the ribs and the crest of the ilium.

Enlargements of the kidney.

**Symptoms.**—The chief symptoms are pain, hæmaturia, cachexia, and enlargement of the kidney. Guillet found that hæmaturia was the first symptom in one-half the cases. The quantity of blood passed may be very small, or so great as to amount to a dangerous hemorrhage. The urine may be red or chocolate colored, and may contain clots of blood or casts of the ureters. Frequent micturition is sometimes an early symptom. In other cases there is no hæma-

turia, the cachexia, emaciation, and tumor being the first symptoms. In younger children the hæmaturia is frequently absent. The kidney is in these cases protected from traumatism. The tumor is sometimes so great as to cause displacement of the organs. In Fürbringer's case the heart was displaced to a situation beneath the clavicle. The abdomen is distended, and the colon is pushed in front of the growth and is indicated by a tympanitic area at one side of the median line of the tumor. On the right side, the tumor appears beneath the liver, and in narcosis can be felt in that situation as a distinct mass. The tumor has an uneven surface. The urine may, in addition to blood, contain histological elements of the growth. This does not occur so frequently in carcinoma of the kidney as in tuberculosis of that organ.

**Duration.**—The progress of the growth is much more rapid in children than in adults. In the former subjects the duration of the disease is from ten weeks to fourteen months (Roberts).

**Diagnosis.**—In children, while the diagnosis of a morbid growth of the kidney can be made, it is not possible to differentiate between the symptoms of carcinoma and those of sarcoma. It cannot be determined, from the symptoms, whether the growth is a simple carcinoma, an adeno-carcinoma, or an adeno-sarcoma. The symptoms of a malignant growth of the kidney are pain, hæmaturia, tumor, and cachexia. A cyst of the kidney may be confounded with a malignant growth. Cysts are congenital, and as a rule bilateral. This is also the case in hydronephrosis. In the latter condition extraperitoneal puncture of the tumor may give a fluid with urine constituents. In carcinoma of the kidney, puncture for diagnostic purposes is not devoid of danger.

### **Tuberculosis of the Kidney.**

Tuberculosis of the kidney is rarely if ever primary. Senator asserts that it never occurs as a primary lesion. There are pathologically two forms—the miliary and the cheesy. The miliary form is more frequent in children, the cheesy in later life. In the miliary form, the kidney tissue is the seat of an eruption of miliary tubercles. In the cheesy form, tuberculous nodules may entirely replace the substance of the organ. The cheesy form is as a rule secondary to tuberculosis of the genitals—the epididymis in boys and the tubes in girls. The symptoms do not differ materially from those of the same condition in adults. In the miliary form there are no symptoms. In the cheesy variety there are dysuria, strangury, vesical tension, pain in the region of the kidney, emaciation, and fever. The urine contains albumin, blood, epithelium, and pus cells, and is acid in reaction. Tubercle bacilli are sometimes found.



The **diagnosis** rests on the presence of tubercle bacilli in the urine, an enlarged palpable kidney, hæmaturia, and tuberculosis of other organs—the genitals or the lungs.

#### **Treatment of New Growths of the Kidney.**

The treatment of new growths of the kidney is within the province of the surgeon. The congenital cysts are of scientific interest only. If there is reason to believe that there is congenital hydronephrosis which is unilateral only, surgical interference is indicated. Sarcomata and carcinomata should be treated surgically if there is reason to believe that there are no metastases in the liver or elsewhere. Tuberculosis of the kidney is treated more from a general standpoint. If there is tuberculosis elsewhere, palliative treatment alone must suffice. Isolated tuberculosis of one kidney is a rare condition which necessitates extirpation of the organ. If it is impossible to determine the proper treatment, an exploratory operation is indicated.

#### **PYELITIS—PYELONEPHRITIS.**

This is a very rare affection in infancy and childhood. Calculus, tuberculosis, and irritating drugs, are etiological factors. It occurs as a complication of the infectious diseases—scarlet fever, measles, variola, and typhoid fever. It may be caused by infection from a vulvovaginitis or by coli bacteria entering through obscure channels. The inflammation of the pelvis of the kidney may extend to the tissue of the kidney itself. Small abscesses are present in the cortex and there is degeneration of the parenchyma of the kidney.

The **symptoms** consist of fever, of an intermittent or remittent type, with recurrent rigors. The fever may alternate with sub-normal temperatures. The urine is characteristic. It is of normal quantity, and low specific weight, is acid in reaction, milky in appearance, and contains pus, mucus, and albumin. Microscopically there are casts, leucocytes, and bacteria. The bacteria present in recently voided urine include the coli group, *Bacterium lactis*, *proteus*, and *Bacillus pyocyaneus* (Baginsky). It is characteristic of this condition that the pus may suddenly disappear from the urine through occlusion of the ureter on the affected side by calculi, fibrin, or inspissated mucus. It may reappear after a time. Emission and periarticular or intermuscular inflammation are also sometimes present.

**Prognosis** is influenced by the causal agent. Simple coli-pyelitis may retrograde within a few weeks and result in recovery. The

chronic form may last for months. Other forms may lead to irreparable inflammation and degeneration of the kidney.

**Treatment.**—If the condition does not improve under treatment of rest, milk diet, and diuretics, surgical interference may become necessary.

### PERINEPHRITIS AND PARANEPHRITIS.

This condition is rare in infancy and childhood. It is not always possible to determine the cause. If such is the case, the disease is called primary. As a rule, it is secondary to traumatism in the lumbar region, to pyelitis, or to pyelonephritis. It may occur in septicopyæmic processes, and I have seen it follow the infectious diseases, notably scarlet fever. Of 166 cases collected by Nieten, only 26 occurred in children. One case occurred in an infant five weeks old. Gibney's cases ranged from one and a half to ten years of age. The condition is more common on the left side. The pus may burrow behind the liver or spleen, or find its way downward, forming a mass simulating a cold abscess or a perityphlitic abscess. It may perforate into the pelvis of the kidney, the intestine, peritoneum, vagina, or diaphragm, or, may pass along the ileopsoas muscle, and find its way to the hip, and thus appear externally. The kidney may be involved because of its contiguity to the seat of the process. Pleuritic metastases and amyloid degeneration may finally result.

The **symptoms** are usually obscure. The fever is intermittent or remittent. Young children do not as a rule complain of pain. The first intimation of the nature of the disease is the appearance of a swelling in the lumbar region. On bimanual palpation, a tumor which is fixed, tense, and does not move with respiration, is felt deep under the liver, in the region of the cæcum and ascending colon on the right side, or underneath the spleen on the left. Gibney has described these cases and shown how they may be easily mistaken for cases of cold abscess. The thigh of the affected side is held in a condition of semiflexion.

The **treatment** is surgical.

### ENURESIS NOCTURNA AND DIURNA.

This is a functional neurosis of the bladder in which the urine is passed involuntarily, and, as a rule, at night during the first hours of sleep. It may, however, be passed at any time during the night. Some patients have at times no control over the bladder during the day (diurna). Some have enuresis every other night or only once or twice a week, and others suffer from the affection every night.



Cases of enuresis should be differentiated from those in which there is a complete paresis of the sphincter vesicæ. In the latter case the urine simply flows away. These are cases of disease or anomaly of the cord (spina bifida). In enuresis the children may in other respects be in good health. There is frequently a nervous condition. In some cases there is lithiasis or stone in the bladder; in others the etiological factor is *Oxyuris vermicularis*, obstipation, tumor of the bladder, or vulvovaginitis. Cystitis and adenoids have been regarded as causal. In the majority of cases no cause can be found. The condition follows the exanthemata. In boys it usually disappears toward the sixteenth year. I have seen it persist in girls into adult life. Its treatment becomes a very serious problem.

The **diagnosis** is not difficult. The urine should be carefully examined for evidences of lithiasis, cystitis, glycosuria, nephritis, and nematodes, and the bladder for stone. The diagnosis is not made in infants and very young children. In the latter the enuresis is often only apparent. They do not know how to indicate their wants.

**Treatment.**—The urine should be passed before retiring. The patients should take little liquid at the evening meal. The foot of the bed is raised so that the head is slightly lower than the pelvis. The drugs most utilized are ergot and atropine. The former is given in the fluid extract, minims x to xxx (0.6 to 2.0) t. i. d. Atropine is given before retiring in a solution (grain j to ʒij; 0.06 to 30.), a drop for every year of the age (Watson). It is efficient in many cases, but in some children distinctly dangerous. I had one case in which I gave one-half the above dose. The child, five years of age, became slightly delirious and tried to walk out of a window. Many cases will improve, only to be subject to relapses. Marion Sims has shown that enuresis in young girls may be due to an intolerant and very small, contracted bladder. In such cases, he advises gradual dilatation of the bladder by injecting the organ with increasing quantities of an indifferent fluid. If treated in this way, the bladder will eventually retain urine. Most of the cases resist all methods of treatment.

## VULVOVAGINITIS.

(*Urogenital Blennorrhœa.*)

The term vulvovaginitis, or, as it is now called, urogenital blennorrhœa, refers to a gonorrhœal inflammation of the genital tract in children. Before describing the condition it is necessary to refer to catarrhal conditions which are not gonorrhœal, and which are present in the normal state.

Epstein has shown that in the newly born infant there is a

physiological and normal discharge from the vagina. It is an adhesive, mucoid discharge containing epithelial cells and micro-organisms. A few days after birth, this discharge assumes a purulent and, in icterus, an icteric hue. No leucocytes are found in the discharge. In two weeks it ceases and the parts appear normal. This form is not gonorrhœal. A second condition which I have noted in very young children is the result of uncleanness, lithiasis, irritation caused by *Oxyuris vermicularis*, or masturbation. The parts are reddened and eroded, and are bathed with an abnormal serous discharge. There may be a few erosions around the introitus. These cases recover with ordinary care and removal of the source of irritation. Pus is rarely secreted.

A second group of cases occurring in young female children includes those of vulvovaginitis of the simple catarrhal type. These have a scanty or profuse purulent discharge from the vagina, vulva, and urethra, which presents clinically all the features of the specific gonorrhœal group, but is not gonorrhœal. The condition is not of infrequent occurrence. The urethral orifice is swollen and red. The hymen is also swollen and inflamed. The discharge is thin and milky, or greenish and viscid. Microscopically, it shows in the pus-cells bacteria and diplococci in groups, but these do not show either by culture or on staining the characteristics of the gonococci. The history of such discharges is singularly similar to that of the gonorrhœal form. Urination is painful, and the discharge persists even under careful treatment. In one case of this kind I have seen an inguinal bubo. The catarrh, like the gonorrhœal form, affects the urethra, vulva, vagina, and cervix uteri. I am convinced that the discharge is infectious and communicable from one child to another. It may last for months and again recur. Its exact etiology is still unknown. Uncleanness, infection from a vaginal discharge, marasmus, the infectious diseases, or frail health may be the cause.

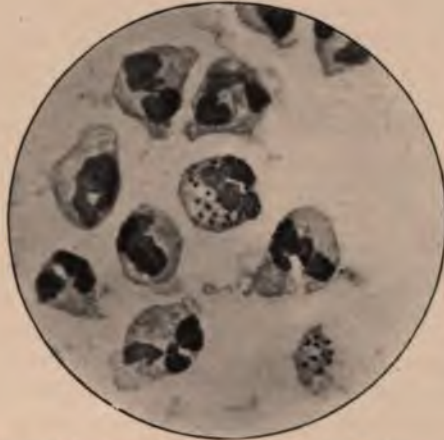
**Urogenital Blennorrhœa.**—These cases have been described by Pott, van Dusch, Spaeth, Cahen-Brach, Epstein, and others.

**Etiology.**—This form may occur in newly born infants (Epstein) or in older infants and children. Epidemics may occur in hospitals (Fränkel). The avenue through which the disease is conveyed is still unknown. It occurs in all walks of life. In some cases there is a history of the child's having slept with the mother. In others, there is no such history. I have sometimes obtained a history of an abnormal attempt at coitus between boys and girls, the boys having suffered at the time from gonorrhœa. Such cases are, however, exceptional. The exciting cause is the gonococcus (Neisser) (Fig. 168). This micro-organism has been found in the discharges of all these cases, and cultivated (Koplik, Heiman).



**Symptoms.**—There is a thick, viscid, purulent, greenish or yellowish discharge from the vagina, which bathes the parts and dries in crusts on the labia. The opening of the urethra is reddened and swollen. There is a discharge from the urethra. Micturition is painful. In some cases there are slight swellings of the inguinal lymph-nodes. If the speculum which is used for the male urethra is introduced into the vagina (Tuttle's urethral speculum), it is seen that the purulent discharge is present in the folds of the mucous membrane of the vagina. The cervix uteri also contains a drop of pus. Thus the whole genital tract is involved. Some children complain of pain over the lower part of the abdomen. On examination, this is found to be pelvic, and is probably due to inflammatory reaction of the tissues about the uterus and vagina.

FIG. 169.

Gonococci in vaginal discharge. Cover-glass spread. Photomicrograph.  $\times 1000$ .

**Complications and Course.**—The course of the disease is quite tedious, and may occupy eight weeks, three months, or more. The discharge may abate, only to return in its original severity.

Peritonitis has in rare cases been reported as a complication of this form of vulvovaginitis. It may prove fatal. Hunner and Harris recently reported a fatal case in a girl ten years of age. They collected 5 other cases from the literature occurring in children. Pelvic peritonitis occurred in 2 of my cases with the usual signs of pain and fever. Both cases made a good recovery.

Hartley and I have reported cases of arthritis complicating vulvovaginitis in children. My cases occurred in the first and second weeks of the disease. In one case, only one joint was affected; in another, two. Both recovered without suppuration.

Gonorrhœal conjunctivitis may result from careless infection of

the eyes. I have had only 2 cases in which the patients complained of præcordial pain. In neither were there active symptoms of endopericarditis, but there is no reason why it might not occur in children, as in adults.

Sänger at one time traced a connection between sterility in later life and attacks of this disease in childhood.

**Treatment.**—Prophylaxis is of great importance. A child affected with the disease should not be allowed to sleep with other children. The toilet appliances should not be used by other children. The parents should be carefully enlightened concerning the infectious nature of the affection and the great danger to the eyesight should infection of the eyes occur. The hands of the patient should be kept scrupulously clean. In institutions the patients should be strictly isolated. The vulva should be kept covered with a pad of absorbent gauze, and a diaper should be worn over this to prevent the discharge from soiling the clothes. In the acute stage, the vagina should be irrigated with a glass catheter or a Skene urethral catheter twice daily. The solution should be at a temperature of 108° F. (42.2° C.). The irrigating solutions should be either a 2 per cent. solution of acetate of aluminum or a 1 : 2000 or a 1 : 500 solution of nitrate of silver. If the silver or aluminum solution is irritating, a simple saturated solution of boric acid may be used. In the subacute stage the vagina is painted once daily with a 5 or 10 per cent. solution of nitrate of silver. A Tuttle urethral speculum is used for the purpose. If the child is intractable, it is impossible to do this without the use of an anæsthetic, which, however, seems scarcely justifiable. I have cured these cases with rest in bed and irrigations. I have tried the bougie treatment and the protargol and permanganate of potassium irrigations, but have found the treatment above described preferable.

### URETHRITIS IN MALE CHILDREN.

Simple urethritis of the anterior portion of the urethra occurs in infants and young children. It is caused either by unnatural interference with the parts or infection. It is not gonorrhœal. The meatus is slightly red or the parts are agglutinated with dried pus. On pressure, a drop of pus exudes from the urethra. There is ardor urinæ, due to a slight fissuration of the meatus. The affection is easily cured by attention to cleanliness. An alkali, such as citrate of potassium, is given in very small doses, to alleviate the ardor urinæ.

Gonorrhœa occurs in male infants and boys, and is the result of direct infection. The symptoms are much the same as in adults, except that, as a rule, there are no complications. Balanoposthitis and lymphadenitis may occur, also epididymitis, and rarely orchitis. Bokai reports cases of stricture.



**CYSTITIS.**

Cystitis is not very common in infants and children. Attention has recently been drawn to this affection by Escherich, who reported several cases of cystitis in young female children caused by coli bacteria.

**Etiology.**—Barlow classifies cases of cystitis as follows:

(a) Chemical cases, caused by drugs.

(b) Bacillogenous cases, caused by the tubercle bacillus, the *Urobacillus liquefaciens*, *Bacillus coli communis*, and the typhoid bacillus.

(c) Coccigenous cases, caused by the gonococcus, staphylococcus, streptococcus, and various diplococci.

Of all the micro-organisms mentioned, the *Bacillus coli communis* is the most frequent cause of cystitis (Melchior).

The direct exciting causes of cystitis in children, as in the adult, are cold, catheterization, or calculi. It may follow urethritis, vulvitis, or may complicate the infectious diseases—scarlet fever, typhoid fever, and diphtheria. The changes in the bladder are, as in the adult, swelling and hyperæmia of the mucous membrane. In chronic cases there are thickening of the rugæ, ulcerations, hemorrhages, and the formation of false membrane (diphtheria).

**Symptoms.**—The symptoms are deep-seated pelvic pain, a desire to pass urine, and, frequently, pain in urination. There may be slight fever. The urine is passed in small quantities, is cloudy, and contains flocculi and shreds of mucus and muco-pus. There is sometimes a sediment of creamy consistency. The urine may contain blood and pieces of false membrane (diphtheria). In tuberculous cases, there are tubercle bacilli in the urine.

The cases may be acute, lasting only a week or two, or may be chronic, and last for months. In the variety caused by the coli bacteria (Escherich) the urine is acid. It may be acid in other acute forms. In the chronic tuberculous cases the urine may be alkaline and contain crystals of triple phosphates. The tendency in acute cases is toward recovery. Complications, such as pyelitis and peritonitis, may occur.

**Treatment.**—If the symptoms are acute, the child is put to bed, and the bladder washed out with a solution of creolin. Salol, grain ij (0.12), is given three or four times daily. Urotropin, grain iij (0.18), is of great utility in the ammoniacal forms of cystitis. Alkaline waters (Vichy) are given freely and the bowels are kept open with alkaline salts. In convalescence an alkali, such as citrate of potassium, given in grains v (0.3) t. i. d., is beneficial.

In chronic forms saccharin is sometimes the only drug that will give relief. Grains ij (0.12) t. i. d. may be administered to a child of six years with safety.

## LEADING AUTHORITIES REFERRED TO IN CHAPTER XIII.

- Abl, J. A.* : "Floating Kidneys in Children," Jour. Amer. Med. Assoc., 1901.
- Baginsky, A.* : "Ueber Pyelonephritis im Kindesalter," Deutsch. med. Wochen., 1897.
- Barlow* : "Beitrag zur aetiol. and Cystitis," Arch. f. Dermat., 1893.
- Bockenheimer* : Die Kongenitale Cysteniere, Dissert., Würz., 1897.
- Comby, J.* : "Floating Kidney," Trans. British Med. Assoc., 1898.
- "Lithiase chez les Enfants," VI. Verhandl. gesell. Kinderheilk.
- Churchill, F. S.* : "Cyclical Albuminuria," Trans. Amer. Ped. Soc., vol. xiii.
- Cohen-Brach* : "Urogenital Blennorrhœa," IX. Verhandl. gesell. f. Kinder., 1891.
- Delafield, F.* : "Bright's Disease," Amer. Jour. Med. Sciences, 1891.
- Escherich, T.* : "Ueber Cystitis bei Kinder," Mitt. aus der Verein der Aerzte, Steiermark, No. 5, 1894.
- Fordyce* : "Cases of Urinary Infection with Bact. Coli," Jour. Cutan. and Gen.-Urin. Dis., 1893.
- Freemann, R. G.* : "Nephritis in Influenza," Trans. Amer. Ped. Soc., vol. xii.
- Hartley, F.* : "Gonorrhœal Rheumatism," New York Med. Jour., 1887.
- Heiman, H.* : "Vulvo-vaginitis," New York Med. Rec., 1895-1898.
- Heineke* : Maligne Nierengeschwulste in Kindersalter, Dissert., München, 1897.
- Hunner and Harris* : Acute General Gonorrhœal Infection, Bull. Johns Hopkins Hospital, vol. viii., No. 135.
- Jacobi, A.* : "Primary Sarcoma of the Kidney," etc., Trans. Internat. Med. Cong., Copenhagen, 1884.
- "Floating Kidney" in Therapeutics of Infancy and Childhood.
- Koplik* : "Urogenital Blennorrhœa," Jour. Cutan. and Gen.-Urin. Dis., 1893.
- "Arthritis Complicating Vulvo-vaginitis," New York Med. Jour., 1890.
- Lewi, E.* : "Sarcoma of the Kidney," Arch. of Ped., 1896.
- Melchior, M.* : Cystitis u. urinfection, Berlin, 1897.
- Morse, J. L.* : "Renal Complications of Acute Enteric Origin," Trans. Amer. Ped. Soc., vol. xi.
- Pott* : Jahrb. f. Kinderheilk., 1883.
- Rachford, B. L.* : "Albuminuria as a Lithæmic Manifestation," Trans. Amer. Ped. Soc., vol. x.
- Singer* : Die Tripperansteckung beim weib, Leipzig, 1889.
- Senator* : "Die Erkrank. der Nieren," Nothnagel's Spec. Path. und Therap., Bd. xix.



## CHAPTER XIV.

### DISEASES OF THE SKIN.

THE skin of the infant is exceedingly delicate in structure. After birth there is a physiological condition of desquamation, as a result of which the skin is very sensitive to a traumatism which in older children would be considered slight. In the newly born infant, such is the delicacy of the structure of the skin that infection may occur when no lesion of continuity is apparent (cryptogenic). A rapid examination of the skin is the first step in making a full physical examination of an infant or child. The surface is first inspected from a distance, the color and the presence or absence of an eruption being noted. It is of the first importance to decide whether an eruption is acute or connected with constitutional taint (syphilis). An eczema may in a syphilitic infant have certain characteristic variations of color which will at once lead the examiner to suspect constitutional disease. A familiarity with acute eruptions (exanthematic) is essential. These must be diagnosed or excluded before any treatment can be inaugurated. Forms of œdema must be differentiated from sclerema and myxœdema, and indurations of the skin from elevations. A papule may be elevated but not indurated. Since the skin of infants and children is exceedingly delicate, it will show indurations more distinctly than that of the adult.

**The Care of the Skin.**—Stretching or harsh manipulation of the skin of infants will tear or traumatize it. Irritating soaps should not be used. The drying of the skin should be carried out gently. The skin in the groin and axilla should not be unduly stretched lest rhagades or fissures result. In powdering the skin, a fresh pledget of absorbent cotton should be used as a powder puff, and all the excess of powder blown off, lest caking result. In some infants the wearing of flannel or wool next to the skin causes irritation and eruptions of different varieties. Such infants should wear a very fine cambric or linen garment next the skin, and over this the woollen shirt.

### ECZEMA.

Eczema is a very common affection in infancy and childhood.

Some infants, otherwise in apparent health, suffer at times from a very mild eczema of the face, which appears chiefly on the cheeks, but which may also be present on the chin, forehead, and ears. The

infants do not seem to suffer much, except that they scratch the eruption. The eruption is local. It may improve without treatment, but if there are conditions of traumatism and infection, it will grow worse. It is rarely moist, but, if scratched, it will bleed, and fissures or ulcers with bloody crusts will form.

Another form of eczema is pustular and vesicular. The skin of the face has a red, angry look. Here and there, patches of skin are covered with scabs; in other areas the skin is moistened by a serous or seropurulent exudate. This eczema is usually also present on the hands and arms. If the malady has existed any length of time, there is considerable thickening of the skin of the hands. The head and scalp may be affected.

Eczema is sometimes general. On the face, it is general and pustular; on the body, there are both the squamous and the pustular forms with all the various gradations between. There are crusts, rhagades, and areas of superficial loss of tissue.

The infants scratch and are uneasy and restless at night, but the general health is excellent and the appetite and digestion are good. The weight increases. If the eczema is general, the infants sometimes become puny. They scratch the eruption, constantly causing the surface to bleed. The body is sometimes one raw, suppurating surface. The lymph-nodes connected with the affected surface are enlarged. Such enlargements should be differentiated from those of pyogenic origin.

A very troublesome form of eczema is the impetiginous or pustular variety. The pustules burst and leave the surface covered with dried crusts of pus. This form may affect any part of the body. Of especial interest, and in a class apart, is the so-called impetigo faciei contagiosa. This is a contagious pustular eczema. It affects by predilection the upper lip and the *alæ nasi*. The pustules break down and leave dry crusts of a golden-yellow color. The anterior nares may be blocked up by these crusts. This variety of impetigo may in children spread over the whole surface and the extremities. I have seen it affect several children in a family. There can be very little doubt as to the infectious and contagious nature of the malady. Eichstedt, Lustgarten, and others have, with cocci obtained from the pustules, succeeded in inoculating the malady on the human subject.

Intertrigo (eczema intertrigo) or erythema intertrigo is one of the forms of erythema which develop by maceration into an eczema. Intertrigo is found in the folds of the neck, axilla, and groin, in well-nourished, rather obese infants. It is at first acute, but may become chronic. There is at first a slight redness of the folds of the skin (erythema). If through neglect the epidermis is allowed to macerate, excess of secretion results and the collected secretions decompose; the surfaces may become eroded, and ulcerations result. In some cases there are lineal ulcers in the groin. In others, the ulcers may



become coated with a pseudomembrane. In rare cases actual necrosis of tissue results. Some anæmic infants present a tendency to rhagade formation, not only in the groin, but also around the anus and lips. The intertrigo may have the color of copper, instead of the bright-red hue of an ordinary eczema. In such cases there is always a possibility that the intertrigo may be of syphilitic origin. If there is no great panniculus of fat, and if with the intertrigo there appear erythema and fissures between the toes, and glossiness of the skin on the plantar surface of the feet, there are additional grounds for assuming that there is a syphilitic element. Intertrigo, like other skin eruptions, may be accompanied by enlargement of the lymph-nodes leading from the region affected. In obese infants, the umbilicus may also be the seat of eczema, which results from the accumulation and decomposition of secretions.

Seborrhœa capillitii is an eruption on the scalp of infants and children, which is classified by Unna as a form of eczema. The scalp is covered with a coating of yellow or discolored sebum, which consists of fat, desquamated epithelium, and hair. If allowed to accumulate, it is sometimes of considerable thickness and may be detached from the scalp. It then leaves a slightly reddened surface, which may bleed. In a short time the scalp may become glossy, and a new layer of the fatty secretion may form. This process may continue until the second or third year. This seborrhœic eczema has sometimes a cheesy odor.

Seborrhœa of the umbilicus has been mentioned. In infants and children there may also be seborrhœa of the prepuce. There are, in neglected cases, secretion and aphthous ulcerations of the folds between the glands and the prepuce and in the folds of the prepuce.

Of great interest to the physician is a form of intertrigo or eczema found on the buttocks and between the nates of infants. It occurs in infants who are not kept dry and whose urine decomposes easily if the diapers are not changed frequently. This is a most troublesome form of eczema. The nates are at first red, the skin then becomes glossy and brittle, and there may be extensive desquamation of the surface. This form of eczema or intertrigo may disappear under treatment, only to return if precautions as to cleanliness and dryness are not observed. Some of the children suffer from enuresis, and contract the affection through maceration of the skin by the decomposed urine, or from unclean diapers.

The **etiology** of eczema is still obscure. The conditions in infancy and childhood are favorable to the development of skin affections. The delicacy of the skin, its constant exposure to dirt and to irritants of all kinds, and changes of temperature, are etiologically important. All the children of a family may suffer from eczema. In such instances, there is a real hereditary tendency to the disease. The parents are sometimes similarly affected.

The influence of diet in causing eczema is not yet understood, but some authors are firmly convinced of the deleterious effects of certain articles of food. I have known urticaria to be caused by eating oatmeal and fruits, such as strawberries, and urticaria may be the beginning of eczema. In most cases eczema cannot be attributed to articles of diet. It is possible that in certain children the processes of metabolism are at fault. Though it has not been proved that all eczema is of an infectious character, there can be but little doubt that many forms are caused by the deleterious action of micro-organisms on the skin (Unna). In favor of this theory is the fact that in many parasitic skin affections eczema is an accompanying condition.

The **treatment** of eczema is exceedingly difficult. The external causes of irritation should be immediately removed. Attention to cleanliness is alone sometimes sufficient to cure an eczema. If woollen clothing is irritating to the skin, a substitute should be found and cotton or cambric should be worn underneath the wool.

The diet should be regulated. This is not an easy task, since it is not known what articles of diet produce eczema. If the infant is at the breast, the diet of the wet-nurse and her daily habits should be regulated. Even when the nurse takes simple food, and the milk is flawless, the infant may suffer from eczema. If the nurse is addicted to the use of beer, or vegetables, such as asparagus, the quality of the milk may be affected. The diet of a wet-nurse should not be changed more often than is necessary, else the secretion of milk may cease. If the wet-nurse has a rheumatic or gouty tendency, it is wise to change nurses. On the other hand, an infant may be overfed and excessively fat. In that case the intervals between nursing should be lengthened. To attempt to change the percentage of fat in the milk is not only of questionable utility, but is not always feasible. If the nurse is constipated, the bowels should be regulated, and she should take abundant exercise. Artificially fed infants are still more difficult to manage. If the infant is thriving, interference with the food percentage is not always clearly indicated. Artificially fed infants may also be overfed or the percentage of fat or proteids may be too high. There may, however, be eczema even when the composition of milk is proper for the infant, age and weight being taken into consideration.

If there are acidity of the stomach, excessive flatus, constipation, or green stools, regulation of diet is of more practical utility. In such cases it may cause the eczema to diminish. If there is stomach acidity, an alkali (lime-water) should be added to the food. Constipation and flatulence should be remedied. If the infant passes urine loaded with urates to such an extent as to cause a red deposit on the diaper, small doses of bicarbonate of sodium should be administered and lime-water should be mixed with the food.



Changes of diet are helpful only in those forms of eczema which are either general or disseminated over different parts of the surface. Seborrhœa and intertrigo are purely local affections, and are not influenced by changes of diet.

Local treatment is chiefly relied upon to improve the condition of the skin. In the acute or subacute forms soothing applications are utilized. The chronic forms are irritated into a state of reaction, and then treated like acute eczema. The treatment of acute local eruptions, such as intertrigo, consists first in keeping the parts scrupulously clean. After the bath the folds of the skin are mopped, dried carefully, and powdered, the excess of powder being blown off. This alone is sometimes sufficient to cure a slight intertrigo. Dusting-powders which contain carbolized preparations irritate the skin. A good powder has the following composition :

R Zinci oxidi . . . . .  $\overline{3}$ iv (16.0).  
Amylum . . . . .  $\overline{3}$ ij (60.0).—M.

Equal parts of zinc and starch powder make an equally good powder. These ingredients should be ground to an impalpable powder. In the severer forms of intertrigo, the parts should first be anointed with ointment having the following composition :

R Resorcin . . . . . gr. ij-iv (0.12-0.24).  
Adeps benzoinati . . . . .  $\overline{3}$ j (30.0).  
M.—The lard should be washed.

The ointment should be removed from the folds of the skin with a pledget of lint. The skin after being thus left in a slightly greasy state is powdered, the excess of powder being blown off. If there are lineal ulcers in the groin, they should be lightly touched once a day with a 2 per cent. solution of nitrate of silver, to promote granulation. The ointment should then be applied with a small piece of lint.

In squamous eczema, which is a red or pustular eczema of the face, scalp, and hands, the first question that arises is whether the patients should be bathed. An infant should be kept clean, and there is only one satisfactory method, and that is the bath. If there is eczema of any part of the surface, the bath water may be liberally impregnated with bran. A gauze bag filled with a measure of bran is put into the bath and the bag squeezed until the water becomes turbid. If a minute quantity of bicarbonate of sodium is added to a bath prepared in this way, the effect on general eczema is decidedly soothing. The skin is gently dried after the bath and powdered. If the whole trunk is involved, it is best that the parts of the surface should be treated in succession. The face or an arm is covered with an ointment applied by means of a piece of lint, or the ointment is simply rubbed on the skin after the bath. It is not feasible to wrap the whole body in lint and ointment; with certain drugs, such as

resorcin, absorption would occur. The ointments should be applied after the crusts and pustular accumulations have been removed. All ointments should be made up with washed benzoinated lard. Vaseline is very irritating to some forms of eczema. Of the emollient and soothing ointments, diachylon, zinc, and bismuth hold a leading place. A very good ointment for general use in rhagades and squamous eczema is the following, which is one of Kaposi's formulæ :

R Resin. benzoe pulv. . . . .	℥j (4.0).
Axung. porc. . . . .	℥v (150.0).
Digere cola adde.	
Zinc. oxidat. . . . .	℥j (30.0).
M. et ft. unguentum.	

If made up properly, this is an excellent cosmetic ointment for use in dry eczema. If the skin is dry and thickened, a 1 per cent.  $\beta$ -naphthol applied twice daily will soften it. If this treatment proves irritating, a zinc ointment may be applied immediately afterward.

In many cases of chronic eczema Lassar's paste is beneficial :

R Acidi salicyl. . . . .	gr. xxx (2.0).
Zinci oxidat. }	
Amylum }	āā ℥j (30.0).
Vaselin. . . . .	℥jss (45.0).
M. et ft. paste.	

The following ointment is also excellent :

R Acidi salicylici . . . . .	gr. xv (1.0).
Ung. zinci oxidi . . . . .	℥ij (60.0).—M.

The tar salves and mixtures are useful in cases of chronic eczema in which there is little or no moisture.

R Ol. rusci . . . . .	℥j (4.0).
Ungt. zinci ox. . . . .	℥j (30.0).
M.—For external use.	

or

R Ol. fagi . . . . .	℥ijss (10.0).
Glycerin. . . . .	℥j (4.0).
Ung. diachylon . . . . .	℥jss (45.0).
Balsam. Peru . . . . .	℥p xxx (2.0).—M.

In cases of red eczema of the face, the ointment is best applied on a mask made of lint.

In that form of intertrigo which results from the irritation of urine, the condition of the diaper is frequently the chief source of trouble. It is often damp or too thin. As a result, whenever the infant passes urine, the diaper becomes saturated with it and decomposition takes place. A piece of absorbent gauze as large as the diaper should be placed next the skin, and renewed whenever it becomes moistened. The skin is dried and the ointment applied on the gauze. Intertrigo is quickly cured by this treatment.



### Treatment of Seborrhœa of the Scalp.

The accumulated sebum is moistened with oil, or a piece of lint moistened with olive oil or any indifferent oil is applied at night. In the morning the crust of sebum will have softened sufficiently to allow of its removal with green soap and water. After the parts are well cleaned, a salicylated ointment, 0.5 to 1 per cent., is applied daily. The ointment should be sparingly applied in order that it may not irritate the parts. Seborrhœa should be treated for some time after it is apparently cured, or it will return. In older children who have abundant hair, the seborrhœa accumulates at the roots and the scalp has an odor. The head should be thoroughly shampooed once a week; after the shampoo, an exceedingly small quantity of cosmetic hair oil should be applied to the scalp once a day.

### ERYTHEMA MULTIFORME.

(*Erythema Nodosum*; *Erythema Exudativum*.)

*Erythema exudativum* is divided into two forms. The acute form includes erythema multiforme and nodosum, and is an acute infectious disease (Lewin). The exudative form occurs frequently in infants and children. Of 40 of my cases, 10 were under two years of age. The form of erythema known as erythema nodosum begins with general malaise and sometimes with fever, which may be quite high. There is pain in the joints and over the areas affected. These areas are raised and are purple or bluish; the skin is tense and the parts affected are very painful. The nodes vary in size. They first appear chiefly on the extensor surface of the tibiæ. The extremity sometimes looks as if it had been beaten. This form of erythema is perhaps allied to hemorrhagic diseases, such as peliosis. In a case of peliosis rheumatica which I saw recently there were erythematous and painful nodules on the hands. Anti-toxin may cause exudative erythema. As is well known, such toxic infection also involves the joints. The symptoms are fever, pain in the joints, and extensive erythema nodosum. I have seen such a case in a subject, who had received an immunizing injection. Within six hours, the legs, knees, and thighs were swollen and the seat of this peculiar erythema.

French writers speak of the frequency of cardiac disease in erythema nodosum, and of its relationship to rheumatism. I have carefully studied 40 cases for signs of cardiac disease, and could find only 3 cases with systolic murmur at the apex. I have recently seen 2 others. In my opinion, true endocarditis is not a very common complication of erythema nodosum. In only one case did the

murmurs appear to be serious. The disease lasts only a few days, but there may be relapses.

The second form of chronic erythema resembles the acute form. The nodules are flat and deep, and are not raised much above the surface. They appear chiefly on the lower extremities of badly nourished children. They are less painful than in the acute form. After a time they disappear leaving no sign of their presence.

**Treatment.**—Cases of erythema of the acute form are treated with sodium salicylate and a diet of milk at first, fruit-juices and beef-juice being given later, and local applications of oil of wintergreen to the painful areas.

### FURUNCULOSIS.

(*Folliculitis Abscedens or Perifolliculitis Abscedens.*—ESCHERICH.)

This affection of the skin is very common in infancy and childhood, and occurs chiefly in badly nourished, marantic babies, who suffer from gastro-enteric and pulmonary infections. The disease is due to an invasion of the deeper layers of the skin by staphylococci. These have been found in the pus and in the sweat and sebaceous glands of the skin (Escherich). In the mild forms of furunculosis there are one, two, or more furuncles on the forehead, scalp, occiput, and neck. Sometimes the furuncles are large and the skin is riddled with them, but as a rule they do not communicate with one another. In aggravated cases, furuncular abscesses occur on the trunk and on the upper and lower extremities. When the furuncles or boils become very numerous, they play a leading rôle. Many children in institutions succumb to this affection. The condition closely resembles a form of sepsis.

The **treatment** of these cases is simple. I have administered alkalies, such as bicarbonate of sodium, internally. The effect on the general process is excellent. I have also given sulphide of calcium in grain  $\frac{1}{2}$  doses (0.03) with good effect. The infant is bathed in bran daily. Too many of the abscesses should not be opened at once, and they should not be opened until they point and the skin over them becomes reddened. If they are opened earlier, the results are not so good. After the abscesses are opened, the pus is expressed and a moist dressing applied. The abscesses heal easily. As in other septic affections, the patients should be stimulated and carefully fed. Small furuncles appearing only on the face need not be opened. The application of a 2 per cent. salicylated ointment twice daily softens the pustules and causes the contents to be discharged.



**SUDAMINA.**

(*Miliaria Alba; Miliaria Rubra.*)

Sudamina is an affection occurring in infants and children during very warm weather. In the form called *miliaria alba* the epidermis at the openings of the sweat-glands is raised by a minute serous exudate and small vesicles are formed. There is no inflammation of the skin. In a second form, the same process takes place, with the presence of a minute focus of inflammation and redness at the opening of the glands. Some of the vesicles are pustular. There are also numerous papules of eczema. There is a slight infection of the skin about the opening of the sweat-glands. Both these conditions are irritating, but in no way serious. The skin should be kept scrupulously clean and dried with powder. Woollen fabrics should not be worn next the skin. If the condition becomes severe, bran baths and a bland zinc or diachylon ointment should be used. Sudamina of both varieties are met with in scarlet fever dermatitis.

**DERMATITIS EXFOLIATIVA.**

(RITTER VON RITTERSHAIN.)

This affection is peculiar to the newborn infant. Ritter in 1878 described an epidemic. In 1895, Escherich published an account of a small local outbreak in Gratz.

**Nature and Etiology.**—It was first suspected by Ritter to be one of the septic infections of the newly born infant. His view has lately been supported by Escherich.

**Occurrence and Symptoms.**—The disease appears from a few days to two weeks after birth. It usually occurs in poorly nourished infants, but may affect apparently healthy infants of normal weight. Boys are more frequently affected than girls. The affection is preceded by the appearance of a diffusely red erythematous or dark swelling of the general surface. The skin is thickened, soft, macerated, and velvety to the touch. The epidermis can be moved on the corium beneath. The pressure of the clothing or bedclothes may also produce this effect. Minute vesicles appear, and coalesce to form larger vesicles or bullæ. Vesicles or bullæ of large size which may be either partly filled with serum or empty are formed. They are never tense, and finally open or tear, leaving the red moist corium exposed. The surface of the body has a beefy-red color, and is covered here and there with patches of dry, adherent epidermis; in other areas, the corium is exposed. There are rhagades at the angles of the mouth and on the trunk. The upper extremities become affected later than the lower ones. Whole areas of the trunk and body are denuded of epidermis. After the vesicles burst and leave

the corium exposed, the epidermal layer of the skin is still adherent in places, while the desquamated skin is rolled up into cord-like masses and hangs loosely exposed. If recovery takes place, the corium becomes covered with a delicate epidermis, which gradually assumes the normal pinkish-white hue. Some cases may run an afebrile, others, a febrile course.

**Course and Prognosis.**—A few of the cases recover. Ritter lost 50 per cent. of his cases, and Escherich 90 per cent. The infants may die from the sixth to the tenth day or after the third week, when much of the skin has undergone retrograde changes. The cases may show umbilical infection or lobular pneumonia pointing to the septic nature of the disease.

**Treatment.**—The infants are kept warm by artificial means, such as warming bottles or an incubator. They are not bathed. The skin is protected by the application of bland salves or gauze moistened with a mixture of linseed oil and lime-water (Escherich). Some physicians add a small quantity of salicylic acid to the salves. As soon as the skin has become dry, Lassar's paste and powdered zinc are applied.

### CONGENITAL ICHTHYOSIS.

(*Cutis Sebacea.*)

Ballantyne gives an exhaustive description of this affection, which is really a perpetuation of a foetal condition into post-natal life. The foetal skin has a tendency to seborrhœa. This is apparent after birth, and is evident during infancy as seborrhœa of the scalp. The seborrhœa may affect different parts of the body and may form thin shining scales on the surface of the skin. There may be secondary eczema. The mild forms may, with ordinary cleanliness and the application of bland salves, disappear a few weeks after birth. The form described by Hebra and Kaposi as ichthyosis congenita is an extreme example of the tendency of the foetal skin to the formation of sebum or vernix. The increased secretion continues after birth. The infant appears to be covered with a horny mass which almost envelops it. This parchment-like covering is absent at the mouth, eyes, anus, and on the scalp. The surface is firm and of a yellow or brownish-red tint (Escherich). The hardness and brittleness of the skin render motion painful. The infant is enclosed as if in case-armor. The face has a mask-like expression. The skin is broken in places, especially at the joints. At these fissures the true skin is seen. At the broken spots, the sebum is seen to be composed of lamellæ, from the posterior aspect of which project warty excrescences corresponding to the lanugo and openings of the sebaceous glands. These may be removed from the skin. If the infant lives, the layers of sebum are



thrown off gradually, and the skin is left with a general seborrhœa of the ordinary type. Escherich predicts a favorable course in most of these cases, but some die shortly after birth. Pathologically there is a great thickening of the rete Malpighii; the corium shows no changes; the sebaceous glands are atrophied or the seat of fatty degeneration; the sudoriparous glands are normal. After the layers of horny sebum have peeled off, the skin underneath appears pink or red or shining, and is covered with seborrhœal scales.

The **treatment** consists in the application of emollients and in washing the skin daily or bathing the infant in permanganate of potassium (grains xv (1.0) to the bath water). Salicylic and boric ointments are applied after the baths.

### PEMPHIGUS NEONATORUM.

Pemphigus neonatorum is a contagious infectious disease of the skin occurring in the newborn infant. It has also been observed later in infancy. It usually appears at the end of the first or second week, and affects the whole surface, except the palms of the hands and the soles of the feet. There appear on the surface of the trunk and extremities small and large vesicles containing cloudy serum. These burst and leave a round patch of skin, which dries and is covered with yellowish scales. The vesicles may attain the size of bullæ. They may be discrete or involve the whole body, so that the surface is after a time denuded of the epithelial layer. The disease may in the beginning be confounded with dermatitis exfoliativa. The vesicles may appear in crops; the recurrences may extend over a period of from two to four weeks.

There are two forms, in one of which the disease is mild; in the other, it runs a malignant course, and from the outset large areas of skin are denuded of epithelium by the bursting of enormous bullæ. The infants pass into an asthenic condition, refuse nourishment, and die in a few days. Both forms appear in epidemics. The disease occurs sporadically. The essential cause is still obscure. Strelitz, Demme, Almquist, and Escherich have isolated a white staphylococcus from the serum of the vesicles. Its rôle as an etiological factor is not as yet understood. Escherich is inclined to class this form of pemphigus with other infectious skin diseases, such as the impetigo of Wilson or Bockhart, and folliculitis abscedens, in which certain conditions favor serous infiltration of the horny layer of the skin and extensive desquamation from the corium. He believes the exciting cause to be the pus cocci found in other forms of impetigo. Escherich has suggested the use of the name "*Impetigo Bullosa Neonatorum* or *Infantum*" for this affection.

The **prognosis** is favorable if the process confines itself to the

superficial layers of the skin. If the deeper layers are attacked, abscesses and general sepsis result.

**Treatment.**—Escherich recommends that the affected parts be washed with soap and water, and dressed with a 2 per cent. ointment of white precipitate. Baths are not given. Those who are interested in the epidemiological aspect of this disease will find the monograph of Richter exhaustive.

#### LEADING AUTHORITIES REFERRED TO IN CHAPTER XIV.

*Ballantyne, J. W.*: Antenatal Pathology and Hygiene, Edin., 1902.

*Escherich, T.*: "Diseases of the Skin in the Newly Born," Pediatrics, 1897.

— "Zur ætiol. der Multiplan Abcesse. Folliculitis Abcedens," Münch. med. Woch., 1886, Nos. 51 and 52.

*Richter, Paul*: Ueber Pemphigus Neonatorum, Berlin, 1902.

*Unna, P. G.*: Article "Eczem.," in Handbuch der Hautkrankheiten, Mraček, 1902.



## INDEX OF AUTHORS.

- A** BT, 614, 638  
 Achard, 129, 596, 606  
 Adams, 445, 446  
 Afanasjew, 201  
 Ahlfeld, 73, 84  
 Alix, 47, 84  
 Almqvist, 457, 649  
 Amberg, 334, 355  
 Anton, 276  
 Antonelli, 273  
 Ausset, 462, 467, 588
- B** ABES, 37, 95, 289, 290,  
 304, 392, 584, 605  
 Babinski, 257  
 Backhaus, 58, 84  
 Bacon, 601  
 Baginsky, 107, 109, 113,  
 145, 154, 206, 216, 235,  
 317, 326, 392, 459, 479,  
 540  
 Ballantyne, 648, 650  
 Banti, 392  
 Barfurth, 48  
 Barker, 113  
 Barlow, 446, 565, 593, 588  
 Barlow (Ger.), 637, 638  
 Bartels, 144, 152, 156, 624  
 Barthez, 239, 251, 610  
 Bauer, 458  
 Baumgarten, 241, 243  
 Bäumlcr, 555  
 Beck, 108, 355  
 Bednar, 277, 436, 458  
 Behring, 235, 276  
 Bein, 197  
 Benecke, 555  
 Berggrün, 579, 587  
 Berkeley, 491  
 Bernabei, 282  
 Bernard, 320, 564  
 Bernheim, 287, 469  
 Bertin, 88  
 Besnier, 238  
 Biedert, 66, 73, 355  
 Billard, 458  
 Billings, 383  
 Birch-Hirschfeld, 236, 243  
 Bishop, 304  
 Blackader, 242, 276, 304  
 Blanchard, 552  
 Blaunberg, 309  
 Blumenfeld, 599, 605
- Blumer, 276  
 Bockenheimer, 638  
 Böhm, 292, 588  
 Bokai, 295, 304, 617  
 Boltze, 532  
 Bonnard-Favre, 432  
 Booker, 208, 316, 328, 355,  
 432  
 Bordoni-Uffreduzzi, 191  
 Borger, 355  
 Botkin, 610  
 Bouchard, 548  
 Bouchut, 624  
 Bourdillot, 154  
 Boussage, 235  
 Bovaird, 242  
 Bowditch, 414, 432  
 Bretonneau, 36, 206  
 Breyer, 154  
 Brieger, 108  
 Briquet, 476  
 Budin, 54, 55, 84  
 Bugge, 243  
 Bullinger, 241  
 Bürkel, 593
- C** AHEN-BRACH, 634,  
 638  
 Caillé, 240, 499  
 Camerer, 53, 55, 66, 73, 84,  
 615  
 Cantani, 42, 84  
 Carr, 593  
 Carstanjen, 572, 593  
 Celli, 276, 330  
 Cesaris-Demel, 491  
 Cestan, 432  
 Chapin, 84, 521  
 Charcot, 523, 545, 555  
 Chaterinkoff, 311  
 Chauffard, 610  
 Cheadle, 588, 593  
 Churchill, 638  
 Chvostek, 475, 481, 548  
 Clark, 479  
 Cohnheim, 102  
 Comby, 614, 638  
 Conrad, 84  
 Contaret, 305  
 Cornet, 236, 239, 276  
 Cornil, 392  
 Cotton, 552, 555  
 Coult, 551
- Councilman, 191, 208, 219  
 276, 469, 620  
 Courmount, 606  
 Crandall, 490, 588  
 Cruse, 111  
 Cutter, 289  
 Czaplewski, 201  
 Czerny, 97, 305, 308, 355  
 432, 621
- D** ANA, 507  
 Decaisne, 60  
 Deichler, 201  
 Delafield, 191, 392, 443, 459,  
 500, 501, 619, 620, 638.  
 Demme, 251, 568, 611,  
 613, 618, 649  
 Dench, 604, 606  
 Dennig, 252, 258, 276  
 D'Espine, 165  
 Doane, 71, 84  
 Döderlein, 627  
 Doehle, 35  
 Dolega, 564  
 Donkin, 551  
 Doremus, 554  
 Dreschfeld, 446  
 Drewitz, 305  
 Duchenne, 518  
 Duel, 127, 276, 606
- E** BEN, 241  
 Eberle, 268  
 Ebert, 499  
 Ebstein, 553, 628  
 Edwards, 467, 613  
 Ehrlich, 573, 578, 593, 618  
 Eichstedt, 640  
 Elsässer, 484  
 Emmerich, 95  
 Emminghaus, 141, 142  
 Eppinger, 102  
 Epstein, 17, 36, 39, 84, 95,  
 102, 107, 269, 277, 304,  
 335, 355, 483, 553, 575  
 Erb, 479, 481, 518  
 Ernst, 459  
 Erös, 18, 84  
 Escherich, 18, 69, 73, 109,  
 206, 235, 316, 329, 479,  
 484, 532, 541, 581, 593,  
 637, 646  
 Ewing, 383

**F** EER, 18, 31  
Fehling, 28, 84

Felsenthal, 320, 572

Fielder, 276

Filatow, 147, 355

Finger, 266

Finkelstein, 314

Finkler, 432

Fischl, 95, 97, 117, 206,  
272, 276, 432, 479, 532,  
575, 621

Fleischman, 305, 311, 467,  
536

Fletcher-Beach, 560

Flexner, 210, 457

Flindt, 147

Foa, 191

Folger, 613

Folwarczny, 613

Forchheimer, 142, 282, 304,  
574

Forlyce, 638

Förster, 588

Foster, 441

Fournier, 84, 262, 276

Fränkel, 135, 243, 371, 634

Freeman, 70, 638

Freundenberg, 135

Frerichs, 552

Freymanth, 290, 304

Friedeleben, 567, 568

Friedländer, 132, 282

Friedreich, 458

Frosch, 392

Frühwald, 304

Fürbringer, 132, 154, 620

Fürst, 588

**G** AESLER, 276, 606  
Gaillard, 377

Ganghofner, 479, 484

Garnier, 242

Gärtner, 95

Gee, 484, 499, 588, 593

Geissler, 593

Genrich, 104

Gerhardt, 133, 142, 611

Gerster, 344

Gibney, 597, 632

Gibson, 355

Goodhardt, 567

Gowers, 32, 476, 480, 487,  
489, 492, 508, 509, 519, 522

Grandidier, 102

Grawitz, 567

Gregor, 356

Greves, 613

Griffith, 140, 146, 298, 304,  
519, 532

Guillet, 629

Gull, 615

Gultmann, 467

Gundling, 53

Gundobin, 571

Guppen, 571

Gutmann, 548

**H** ADDEN, 499

Hähner, 73

Hammarsten, 65

Hammersley, 572

Hansemann, 555

Harbitz, 442, 443

Harris, 334, 635

Hartigan, 108

Hartley, 551, 635, 638

Hather, 135

Hayem, 570, 593

Hecker, 104

Heermann, 599, 601

Heim, 140

Heinecke, 638

Heirman, 454, 638

Heiter, 458

Heller, 267

Hennig, 116, 610

Henoch, 144, 244, 387, 484,

485, 486, 489, 492, 494,

545, 587, 593, 607, 611,

616, 623, 624

Henschen, 467

Hensel, 201

Herz, 48, 56, 84

Herzfeld, 251, 276

Heubner, 135-136, 191,

235, 306, 309, 315, 356,

471, 545, 552

v. Heukelom, 99

Hildebrand, 140

Hirsch, 194, 317, 504

Hirschsprung, 552, 590

His, 564

Hlava, 548

Hochsinger, 266, 267, 276,

441, 467, 455, 482, 483

Hock, 572, 593

Hodenpyle, 300

Hoff, 618

Hoffman, 207, 372, 520,  
574

Hofmeier, 111

Hofmohl, 116, 412

Holt, 305, 375, 432, 588,

624

Home, 144

Hoppe-Seyler, 58, 65, 618

Horbaczewski, 619

Horsley, 564, 565

Houl, 243

Hudelo, 267

Hunner, 635

Hutchinson, 238, 263, 266

Hüttenbrenner, 99

**I** MMERMANN, 583

Ireland, 532

**J** ABOULAY, 606

Jaccoud, 235

Jackson, 491

Jackson, Hughlings, 472

Jacobi, 78, 239, 448, 467,

490, 541, 544, 548, 551,

565, 566, 567, 593, 614,

628, 638

Jäger, 191, 610, 613

Jahni, 243

von Jaksch, 375, 557, 593

Japha, 579, 593

Jeandin, 165

Jenner, 163

Judassohn, 282

Jürgensen, 119, 131, 144,

152, 376, 385, 432, 494

**K** AREWSKI, 295

Karowin, 305

Kartullis, 329

Kassowitz, 472, 482, 484,

540, 541, 544, 545, 555

Kast, 289

Keating, 467

Kee, 115

Keen, 503

Kehrer, 111, 284

Keller, 78, 79, 84, 321,

375

Kelsch, 578

Kemp, 42, 43, 139, 625

Kessler, 371

Kingdon, 504

Kirmisson, 530, 531, 532

Kissel, 610

Kitasato, 185

Kjelberg, 320

Klebs, 102, 135, 206

Klemperer, 443

Kling, 104

Kmerien, 282

Knapp, 602

Knoepfelmacher, 65, 74,

84

Koch, 594

Kocher, 564

Kockel, 243

Kohts, 272

Kolaczek, 99

Kolb, 584

Kolisko, 208

König, 58, 60

Körner, 606

Kossel, 317, 599, 619

Kundrat, 104

Kurloff, 201

Kussmaul, 473

**L** ABI, 89

Lachman, 628

Lachs, 47

Landau, 104, 117



- Landois, 437  
 Landouzy, 243, 377, 476, 610  
 Landry, 517  
 Lannelongue, 99, 129, 243, 596, 606  
 Lazarus, 446  
 Lebreton, 584  
 Lehman, 243  
 Leichtenstern, 191, 571  
 Lejars, 627  
 Lenbartz, 36, 468  
 Leo, 306  
 Leonhardt, 241  
 Leube, 385, 616  
 Leuckart, 356  
 Levi, 89  
 Levy, 385  
 Lewi, 499, 553, 638  
 Lewin, 430  
 Leyden, 548  
 Libman, 317, 490  
 Lichtenstein, 563  
 Lichtheim, 372  
 Lillienthal, 344  
 Limbeck, 594  
 Litten, 35, 131, 132,  
 Little, 509  
 Llad, 58, 69, 84, 594  
 Lloyd, 572  
 Loeffler, 206  
 Lombard, 197  
 Look, 482, 484, 624  
 Lösch, 334  
 Löschner, 613  
 Lotz, 135  
 Lowit, 578  
 Luschka, 567  
 Lustgarten, 640  
 Luzzatto, 201  
 Luzet, 575  
  
**M**acDONALD, 56, 84  
 Macewen, 276, 532  
 Maggiora, 330  
 Magnus, 499  
 Mallory, 208, 276  
 Manges, 545  
 Mann, 613  
 Marfan, 242, 316, 356  
 Marie, 520  
 Marotte, 304  
 Marshall Hall, 92  
 Mattie, 276  
 Matterstock, 344  
 Maydl, 431  
 Mayer, 219  
 Mayr, 144  
 McBurney, 345  
 McCrae, 579  
 McNutt, 609, 515, 532  
 Meckel, 99  
 Medin, 252, 520  
 Meigs, 84  
 Meinert, 574  
 Melchior, 637, 638  
 Meltzer, 315  
 Mendelsohn, 467  
 Menetrier, 235  
 Merckel, 613  
 Mettenheimer, 613  
 Meyer, W., 315  
 Michaelis, 197  
 Michel, 309  
 Miller, 548, 555, 579  
 Minkowski, 111  
 Miquel, 69  
 Moebius, 21  
 Möller, 588  
 Moncorvo, 551, 555  
 Monti, 36, 68, 69, 109, 204, 206, 266, 536, 552  
 Morse, 31, 276, 320, 356, 542, 555, 579, 594, 621, 638  
 Moschowitz, 117  
 Moser, 432, 470  
 Mosler, 578  
 Mráček, 273  
 Müller, 442  
 Murchison, 119, 608, 611  
 Mussy, 89  
  
**N**AEGELI, 589  
 Nakarai, 243  
 Nansen, 589  
 Naunyn, 111, 491  
 Nauwerck, 589  
 Neisser, 110, 634  
 Netter, 191, 276, 377, 386, 432, 442, 588, 599  
 Neumann, 95, 266, 392, 432, 613  
 Nielot, 304  
 Nicoll, 315  
 Niden, 632  
 v. Noorden, 580  
 Northrup, 150, 201, 216, 235, 242, 244, 392, 432, 594  
 Nothnagel, 251, 344  
 Nottingham, 154  
  
**O**'DWYER, 39  
 Ogle, 491  
 Orban, 308  
 Ord, 564  
 Osler, 119, 258, 366, 490, 491, 511, 532, 545, 560, 563, 594  
 Otto, 570  
  
**P**ACKARD, 302, 443, 467, 547  
 Paltauf, 208  
 Papiewski, 109  
 Park, 235  
 Parrot, 284, 285, 291, 325, 566, 597, 615, 621  
 Pascaud, 117  
 Pasteur, 70, 520  
 Pautz, 308  
 Pavy, 552  
 Pearce, 209, 276  
 Perutz, 377  
 Peterson, 472  
 Petruschky, 290, 304  
 Pfaundler, 305, 314, 356, 468, 532  
 Pfeiffer, 135, 185  
 Polyniere, 392  
 Ponfick, 598, 599, 618  
 Pott, 438, 486, 503, 567, 638  
 Pratt, 432  
 Preobrachensky, 135  
 Preyer, 84  
 Price, 71, 84  
 Pritchard, 356  
 Prudden, 95, 150, 216, 235, 392, 432  
 Putnam, 521  
 Pye-Smith, 491  
  
**Q**UESINER, 392  
 Queyrat, 243  
 Quincke, 468  
  
**R**ACHFORD, 581, 638  
 Rankin, 136  
 Rasch, 598  
 Rauefuss, 190, 442  
 Rees, 284  
 Rehn, 148, 588, 613  
 Reimer, 251  
 Renaud, 276  
 Raymond, 432  
 Ribbert, 268  
 Richter, 650  
 Rieken, 468  
 Rilliet, 197, 239, 251  
 Rindfleisch, 245  
 Ritchie, 315  
 Ritter, 282, 305, 647  
 Riverdin, 564  
 Roberts, 552, 630  
 Robin, 615  
 Roger, 242  
 Rohrer, 628  
 Rokitsansky, 439  
 Roloff, 243  
 Romberg, 131  
 Rosenstein, 628  
 Rosinski, 289, 304  
 Rotch, 191, 305, 532, 571, 594, 606  
 Roux, 297, 235, 578, 629  
 Rybner, 309  
 Runge, 101, 102, 104, 107, 111, 117

- Rütimeyer, 519  
 Ryan, 304
- SACHS**, 488, 491, 504  
 Sahlí, 567, 568  
 Sänger, 187, 532, 638  
 Sansom, 440, 467  
 Santulus, 291  
 Schamshin, 457  
 Schamtyr, 282  
 Schapringer, 21  
 Schiff, 570, 594  
 Schlessinger, 572, 593, 613  
 Schlichter, 59, 84  
 Schlossberger, 619  
 Schmidt, 276  
 Schmorl, 208, 243, 589, 594  
 Schoedel, 589, 594  
 de Schweinitz, 490  
 Sée, 459, 466, 491  
 Seeligmüller, 518  
 Seidl, 261  
 Seitz, 206  
 Senator, 613, 616, 625, 627, 628, 638  
 Sidney, 207  
 Siegfried, 59  
 Silbermann, 111  
 Simmonds, 412  
 Sinkler, 520  
 Slawyk, 147, 276, 470  
 Smith, J. L., 134, 503, 565, 588  
 Snow, 343, 533  
 Söldner, 58  
 Soltman, 109, 467  
 Somma, 272  
 Sonntag, 241  
 Sörensen, 136, 570  
 Soxhlet, 63  
 Spiegelberg, 104  
 Stadelmann, 111  
 Starek, 430  
 Starr, L., 68  
 Starr, M. A., 261, 490, 492, 493, 495  
 Steffen, 251, 252, 366, 432, 434, 442, 447, 458, 459, 460, 608
- Steiner, 476, 478, 491, 588, 614  
 Stengel, 432, 594  
 Steudener, 568  
 Stewart, 614  
 Still, 242  
 Stöhr, 300  
 Störk, 486  
 Strauss, 579  
 Strelitz, 107, 392, 649  
 Strümpel, 509, 520  
 Symington, 358, 435  
 Szemetzenko, 201  
 Szontagh, 65
- TALOMAN**, 215  
 Tardieu, 458  
 Tay, Warren, 504  
 Tenner, 473  
 Terrier, 432  
 Thomas, 140, 152, 160, 492  
 Thomson, 314, 356, 499, 532, 565, 566, 594  
 Thursfield, 276  
 Tizzoni, 548, 584  
 Tödtén, 613  
 Townsend, 490, 565  
 Treves, 339, 340, 356  
 Triesethau, 567, 594  
 Trousseau, 475, 479, 486  
 Tschamer, 135
- UFFELMAN**, 309  
 Ullman, 467  
 Ultzmann, 615  
 Unger, 251  
 Unna, 641, 642, 650
- VALLÉE**, 344, 356  
 VanArsdale, 101, 129, 135, 557, 558, 594, 596, 606  
 Van Dusch, 634  
 Van Panum, 144  
 Van Puteren, 306  
 Variot, 484  
 Veillard, 578  
 Vierordt, 47, 84, 433, 440, 467
- Vigier, 69  
 Vincent, 304  
 Virchow, 209, 529, 544, 567, 574, 577  
 Vogel, 308, 615  
 Voinitch, 461  
 Voít, 204  
 Voorhees, 117
- WALSH**, 290, 304, 463  
 Walshe, 467  
 Watson, 633  
 Wegner, 544  
 Weichselbaum, 191, 386, 443, 446  
 Weigelin, 73  
 Weigert, 102, 235, 243, 245  
 Weil, 385, 467, 545, 555, 610, 613  
 Weiss, 610  
 Welch, 36, 371, 443, 457  
 Welt, 581  
 Wentworth, 470, 575, 594  
 Werhowsky, 457  
 West, J. P., 189, 190, 210, 276, 567  
 West (Eng.), 588  
 White, 58, 69, 84, 432, 594  
 Whitman, 540  
 Widerhofer, 613  
 Widowitz, 571  
 Wilke, 611  
 Williams, 18  
 Winckel, 95, 102, 107  
 Wohlman, 306  
 Wolff, 219  
 Woronichin, 291, 304  
 Wright, 208, 276  
 Wroblewski, 65, 84
- YERSIN**, 207
- ZAMBOLOVICI**, 289,  
 Z 290, 304  
 Zeissl, 266  
 Ziegler, 392, 443, 459  
 Zuppert, 520, 521  
 Zweifel, 555



## INDEX OF SUBJECTS.

- ABDOMEN**, examination of, 25  
 inspection of, 25  
 palpation of, 26  
 retracted, 26  
 shape of, 25
- Abdominal distention, idiopathic, 26  
 typhus. *See* Typhoid fever.
- Abscess of liver, 612  
 periarticular, in scarlet fever, 130
- Accessory sinuses, affection of, in diphtheria, 219
- Achondroplasia, 565
- Acorn-cocoa, 80
- Adenitis, acute, diagnosis of, 558  
 in diphtheria, 221  
 etiology of, 557  
 symptoms of, 557  
 treatment of, 558
- Adenoid vegetations, 298  
 contraindication to operation, 299  
 diagnosis of, 299
- Adhesions, pleural, in empyema, 429
- Agensis corticalis, 509
- Albumin water, 80
- Albuminuria, cyclic, 615  
 postural, 615
- Amaurosis in scarlet fever, 130
- Amaurotic idiocy. *See* Idiocy, amaurotic.  
 idiots, head in, 20
- Amorphism, dental, 281
- Amygdalitis, acute follicular, 300  
 diagnosis of, 302  
 duration of, 302  
 etiology of, 300  
 lymph-nodes in, 302  
 prognosis of, 302  
 temperature in, 302  
 treatment of, 302
- Amyloid degeneration in bronchiectasis, 373
- Anæmia, 572  
 blood in, 574  
 congenital, 572  
 essential, 572  
 lymphatic, 575  
 in malaria, 572  
 pernicious, 587  
 characters of, 587  
 primary, 572  
 pseudoleukæmic, 575  
 the blood in, 576  
 course of, 576
- Anæmia, pseudoleukæmic, etiology of, 575  
 liver in, 575, 609  
 morbid anatomy of, 575  
 occurrence of, 575  
 spleen in, 576  
 symptoms of, 575  
 treatment of, 576
- in scarlet fever, 132  
 secondary, 573  
 symptoms of, 573  
 simple, 573  
 blood in, 574  
 in syphilis, 572  
 in tuberculosis, 572  
 von Jaksch's, 575
- Anæmic murmurs, 574
- Anasarca in nephritis, 622
- Angina, cardiac, in aortic disease, 453
- Ani, prolapsus, 349  
 etiology of, 349  
 symptoms of, 349  
 treatment of, 349
- Antipyresis, 34
- Antitoxin. *See* Diphtheria.
- Antitoxins in mother's milk, 17
- Anus, fissure of, 350  
 diagnosis of, 350  
 symptoms of, 350  
 treatment of, 350  
 spasm of, 350
- Aphasia in pertussis, 204  
 in scarlet fever, 131
- Aphthæ, Bednar's, 283  
 treatment of, 284  
 in diphtheria, 221
- Apnoea, respiratory, in retropharyngeal abscess, 297
- Appendicitis, 344  
 catarrhal form, 344  
 chronic, 348  
 course of, 345  
 diagnosis of, 345  
 fever in, 347  
 forms of, 344  
 frequency of, 344  
 gangrenous, 345  
 mortality in, 348  
 pain in, 347  
 perforative, 345  
 prognosis of, 347  
 symptoms of, 344
- Appendix vermiformis, anatomical landmarks, 344

- Arteritis umbilicalis, 99  
     morbid anatomy of, 100  
     prognosis of, 100  
     symptoms of, 100
- Arthritis deformans, 545  
     rheumatoid, 545  
     in vulvo-vaginitis, 636
- Arthrogryposis, 479
- Ascarides lumbricoides, 353
- Ascites, 25
- Asphyxia in cephalohæmatoma, 116  
     in newborn, diagnosis of, 90  
     morbid anatomy of, 90  
     primary stage of, 90  
     prognosis of, 91  
     secondary stage of, 90  
     symptoms of, 90  
     treatment of, 91  
         Dew method, 92  
         Laborde method, 91  
         Marshall-Hall method, 92  
         Schultze method, 91  
     subsequent to birth, 92  
     symptoms of, 92
- Asthma, thymic, 567
- Ataxia, hereditary, 519  
     course of, 519  
     diagnosis, differential, 520  
     prognosis of, 520  
     symptoms of, 519  
     treatment of, 520
- Atelectasis, convulsions in, 94  
     of the lung, 93  
     symptoms of, 93  
     treatment of, 94
- Athrepsia. *See* Atrophy.
- Atresia, cellular, of labia, 617
- Atrophy, gastro-intestinal, 325  
     etiology of, 325  
     morbid anatomy of, 325  
     symptoms of, 325  
     treatment of, 327
- Auricular-ventricular septum deficiency of, 441
- Auto-infection intestinal, 574
- B**ABINSKI'S reflex, 31, 192  
     in tuberculous meningitis, 257
- Bacillus capsulatus aerogenes, 36  
     infection in hypodermoclysis, 323  
     diphtheriæ, 206
- Bacterial diseases in newborn infants, 17
- Barlow's disease. *See* Scorbutus.
- Basedow's disease, facial expression in, 21
- Bath, daily, temperature of, 44  
     full, 36  
     sponge, 35
- Bednar's aphthæ, 46  
     in septic infection, 96
- Bell's paralysis, 512
- Binder, body, 44
- Birth, injuries during, 113
- Birth palsies, 114, 509, 510  
     clinical symptoms of, 510  
     paralyses, head in, 20
- Blennorrhœa, urogenital, 633
- Blindness in pertussis, 204
- Blood in cretinism, 563  
     in diphtheria, 211  
     in leukæmia, 578  
     in measles, 153  
     in pertussis, 203  
     in pneumonia, 383, 403  
     in rachitis, 542  
     in scarlet fever, 132  
     in simple anæmia, 574  
     specific gravity of, 572  
         in diarrhœa, 572  
         in infectious diseases, 572  
         in newly born infant, 572  
     in syphilis, 271
- Body, length of, in boys and girls, 56
- Bone-changes in hereditary syphilis, 268  
     in scorbutus, 590
- Bones, in cretinism, 564  
     diseases of, 595  
     of head, injury to, 596  
         syphilis of, 596  
     rachitis of, 595  
     syphilis of, 264, 271, 595  
     tuberculosis of, 595
- Botalli, open ductus, 440
- Bowel movements, character of, 57  
     number of, 57
- Bowels, condition of, in examination of infant or child, 19
- Boys and girls, loss of weight in newborn, 53  
     weight and height, 56
- Brain abscess in bronchiectasis, 373  
     hernia of, 117  
     tuberculosis of, 261  
     tumors of, 505, 508  
         etiology of, 506  
         location of, 506  
         symptoms of, 506  
         dependent on location, 507
- Brandt bath, 36
- Breast feeding, signs of insufficient, 57  
     milk, amount taken by infant, 73  
         analysis of, 60, 61, 62  
         bacteria in, 60  
         influence of foods on, 60  
         nurse's, toilet of nipple, 46
- Breasts, caking of, 48, 52  
     care of, 52  
     of newborn infant, 47  
     nipples, care of, 52
- Breathing, forms of, 360  
     bronchial, 360  
     broncho-vesicular, 360  
     puerile, 360
- Breck's infant feeder, 89
- Bronchiectasis, abscess of liver in, 373  
     amyloid degeneration in, 373



- Bronchiectasis, brain abscess in, 373  
 complication of, 373  
 congenital, 371  
 cough in, 372  
 course of, 375  
 deformity in, 373  
 diagnosis of, 373, 374  
 dyspnoea in, 373  
 emphysema in, 373  
 etiology of, 372  
 expectoration in, 372  
 heart disease in, 373  
 laryngeal disease in, 373  
 physical signs of, 374  
 symptoms of, 372  
 temperature in, 373  
 treatment of, 375
- Bronchitis, acute, 362  
 causation of, 362  
 morbid anatomy of, 362  
 physical signs of, 363, 364  
 symptoms of, 362  
 treatment of, 364
- capillary, 362, 364  
 chronic, 366  
 fibrinous, 365  
 complications of, 366  
 diagnosis of, 366  
 etiology of, 365  
 morbid anatomy of, 365  
 physical signs of, 366  
 symptoms of, 365  
 treatment of, 366
- and influenza, 186
- Bronchopneumonia, atelectasis, 93  
 bacteriology of, 392  
 cerebral symptoms of, 396  
 complications of, 399, 401  
 course of, 399  
 and diarrhoea, 399  
 in diphtheria, 215, 219  
 treatment of, 234  
 etiology of, 391  
 fever in, 395  
 gastro-intestinal tract in, 396  
 in measles, 398  
 meningitis in, 399  
 morbid anatomy of, 392  
 occurrence of, 390  
 pericarditis in, 399  
 persistent, 407  
 blood, 407  
 diagnosis of, 408  
 physical signs of, 408  
 symptoms of, 407  
 treatment of, 408
- in pertussis, 203, 397  
 physical signs of, 401, 402  
 prognosis of, 404  
 pulse in, 396  
 in scarlet fever, 398  
 in sepsis, 87  
 signs mistaken for, 402
- Bronchopneumonia, sputum in, 396  
 symptoms of, 393  
 treatment of, 404, 407  
 tympanites in, 396  
 in typhoid fever, 398  
 in varicella, 398  
 vomiting in, 396
- Buhl's disease, 105
- C**ALCULI, renal, 619  
 symptoms of, 619  
 treatment of, 619
- urethral, 616
- Cancrum oris, 289
- Caput succedaneum, 117
- Carcinoma of kidney, 628  
 hæmaturia in, 629  
 of thymus gland, 568
- Cardiac area, conformity of, in children, 24  
 palpation of, 434  
 disease, expression in, 21  
 lesion in erythema nodosum, 645
- Caseinogen, 58
- Catalepsy, 483
- Caustic alkali, lesions following ingestion of, 221
- Cephalohaematoma, 115, 117, 576  
 complications of, 116  
 diagnosis of, 116  
 occurrence of, 116  
 pathogenesis of, 116  
 prognosis of, 117  
 symptoms of, 115
- Cerebral palsy gait in, 32
- infantile, 508  
 diagnosis of, 511  
 etiology of, 508  
 forms of, 508  
 morbid anatomy of, 509  
 prognosis of, 512  
 symptoms of, 510  
 treatment of, 512
- syphilis, hereditary, 272
- Cerebrospinal fluid, 468  
 abnormal, 468  
 in cerebrospinal meningitis, 469  
 in chronic hydrocephalus, 469  
 pressure of, 470  
 sediment of, 470  
 specific gravity of, 468  
 in suppurative meningitis, 469  
 in tuberculous meningitis, 469
- meningitis, 190  
 bacteria in, 191  
 blood in, 194  
 cerebral symptoms in, 192  
 choroiditis in, 192  
 circulation in, 194  
 course and prognosis of, 194  
 diagnosis of, 195  
 endocarditis in, 194

- Cerebrospinal meningitis, epidemic, 191  
 erythema in, 192  
 etiology of, 191  
 hemiplegia in, 194  
 intramuscular abscess, 192  
 joints in, 192  
 morbid anatomy of, 191  
 nasal secretion of, 193  
 occurrence of, 191  
 optic neuritis in, 192  
 paralyses in, 194  
 reflexes in, 192  
 respiration in, 194  
 skin in, 192  
 sporadic, 191  
 symptoms of invasion, 192  
 temperature in, 193  
 treatment of, 196  
 types of cases, 191
- Charcot-Leyden crystals, 334
- Chest, examination of, 22  
 exploratory puncture of, 422  
 fluid in, diagnosis of, 421  
 inspection of, 24  
 movements of, 357  
 palpation of, 24  
 percussion of, 25  
 position in examining, 22  
 wall, resiliency of, 358
- Cheyne-Stokes respiration, 358  
 in cerebrospinal meningitis, 194  
 in tuberculous meningitis, 255
- Chicken-pox. *See* Varicella.
- Childhood, definition of, 17
- Chills, 19
- Chlorosis, 574  
 occurrence of, 575
- Cholera infantum, 316  
 duration and prognosis of, 320  
 hydrecephaloid in, 320  
 nephritis in, 320  
 occurrence of, 320  
 symptoms of, 319
- Chondrodystrophia, 565
- Chorea, cardiac symptoms in, 493  
 classification of, 489  
 diagnosis of, 494  
 disturbances of sensation in, 492  
 duration of, 495  
 frequency and etiology of, 490  
 insanians, 496  
 symptoms of, 497  
 treatment of, 498  
 laryngeal, 493  
 morbid anatomy of, 491  
 motor symptoms of, 492  
 prognosis of, 495  
 and rheumatism, 490, 549  
 in scarlet fever, 133  
 speech in, 492  
 with subcutaneous fibrous nodules, 491  
 symptoms of, 491
- Chorea, temperature in, 494  
 treatment of, 495  
 urine in, 492
- Choroid, tubercle of, in tuberculous meningitis, 259
- Choroiditis in cerebrospinal meningitis, 192
- Chvostek's symptom, 581  
 in tuberculous meningitis, 255
- Cirrhosis of liver, 611  
 etiology of, 611  
 morbid anatomy of, 611  
 symptoms of, 611
- Clothing, infant's, 45, 46
- Colic, 25, 312  
 causes of, 312  
 treatment of, 313
- Coliccolitis. *See* Dysentery.
- Colicystitis, 637
- Colitis contagiosa. *See* Dysentery.
- Colles' law, 266
- Colon, polypoid tumors in, 27
- Colostrum, composition, 58
- Compress, cold chest, 35
- Congenital heart disease, 437  
 classification of, 438  
 diagnosis of, 438, 440  
 dilatation and hypertrophy in, 437  
 murmurs in, 437  
 syphilis, condyloma, 270  
 nasal deformity in, 21
- Conjunctivitis, gonorrhoeal, 635  
 in measles, 153
- Conrad's lactobutyrometer, 61
- Constipation, 57, 335  
 massage in, 338  
 movements in, 336  
 symptoms of, 336  
 treatment of, cathartics in, 338
- Constitutional disease, morbidity, 18
- Conus, pulmonary artery, stenosis of, 439
- Convulsions, 19, 34  
 in atelectasis, 94  
 infantile, 472  
 diagnosis of, 474  
 etiology of, 473  
 morbid anatomy of, 473  
 prognosis of, 475  
 symptoms of, 473  
 treatment of, 475  
 in pertussis, 204  
 in tuberculous meningitis, 254
- Coprolalia, 498
- Cord, umbilical, dressing of, 44
- Cows' milk, bacteria in, 69  
 casein of, 65  
 coagulation of, 65  
 composition of, 64  
 fat in, 65
- Cranial bones, indentations of, 113  
 syphilis of, 273
- Craniorachischisis, 528



- Craniosechisis, 528  
 Craniotabes, 536, 596  
 Cretinism, anaemia in, 574  
   blood in, 563  
   bones in, 564  
   diagnosis of, 564  
   etiology of, 564  
   forms of, 560  
   morbid anatomy of, 564  
   sporadic, 560  
     congenital, 561  
   symptoms of, 561  
   treatment of, 566  
 Cretins, hand of, 563  
 Cries hydrocephalique in tuberculous meningitis, 259  
 Cutis sebacea, 648  
 Cyanosis in atelectasis, 93  
   congenital, 437  
   in Winckel's disease, 107  
 Cystitis, catheterization a cause of, 637  
   complications of, 637  
   in diphtheria, 637  
   etiology of, 637  
   forms of, 637  
   in scarlet fever, 637  
   treatment of, 637  
   in typhoid fever, 637
- D**ACTYLITIS SYPHILITICA, 271  
   Deafness in pertussis, 204  
 Delire de toucher, 498  
 Delorme's operation, 430  
 Denhardt gag, 39, 228  
 Dentition, 277  
   pathology of, 281  
   in rachitis, 278  
   syphilis in, 278  
   ulcer in pertussis, 204  
 Dermatitis exfoliativa, 647  
   course of, 648  
   etiology of, 647  
   nature of, 647  
   occurrence of, 647  
   prognosis of, 648  
   symptoms of, 647  
   treatment of, 648  
 Dew method in asphyxia, 92  
 Diabetes insipidus, 553  
   occurrence of, 553  
   symptoms of, 554  
   treatment of, 555  
   mellitus, 552  
     etiology of, 552  
     occurrence of, 552  
     symptoms of, 552  
 Diaper, care of, 46  
 Diarrhoea, influenza and, 186  
   in scarlet fever, 132  
 Dicrotism, normal, 437  
 Digestive tract, frequency of diseases of, 18  
 Digital exploration of pharynx, 297
- Dilatation of heart, acute, in scarlet fever, 132  
   and hypertrophy in congenital heart disease, 437  
 Diphtheria, adenitis in, 221  
   affection of accessory sinuses in, 219  
   aphthae in, 221  
   bacillus, culture of, for diagnosis, 220  
     systemic infection with, 208  
   bacteriology of, 207  
   blood in, 211  
   cardiac paralysis in, 226  
   complications of, 215  
     treatment of, 234  
   contagion in, 206  
   course and duration of, 215  
   cystitis in, 637  
   diagnosis of, 220  
   etiology of, 206  
   exanthema in, 219  
   feeding in, 226  
   frequency of, 18  
   gastro-enteritis in, 215  
     treatment of, 234  
   general infection in, 208  
   head posture in, 20  
   hemiplegia in, 218  
   laryngeal form, 214  
     treatment of, 227  
   leucocytosis in, 571  
   local forms of, 211  
     treatment of, 226  
   malignant forms of, 213  
   in measles, 154  
   the membrane in, 209  
   middle ear in, 211  
   morbid anatomy of, 209  
   nephritis in, 216  
   occurrence of, 206  
   otitis in, 598  
   paralysis of the heart in, 216  
   prognosis of, 222  
   prophylaxis of, 222  
   pseudobacillus of, 22, 207  
   septic forms of, 212  
   symptoms of, 211  
   thymus gland in, 568  
   treatment of, 222  
     antitoxin in, dosage of, 223  
       eruption, 225  
       effect of, 224  
       mode of injection of, 224  
       time of, 223  
   tuberculosis and, 219  
   and typhoid fever, 219  
   of vulva, 218  
 Diphtheritic paralysis, incoördination in, 31  
 Diphtheroid, 220, 235  
   diagnosis of, 235  
   etiology of, 235  
   occurrence of, 235

- Diphtheroid, symptoms and course of, 235  
treatment of, 235
- Diplegia. *See* Cerebral palsy.
- Diplococcus intracellularis meningitidis, 191
- Ductus arteriosus, patent, 440  
murmur in, 441  
physical signs of, 440
- Dungern capsule bacillus, 95
- Dysentery, 329  
and acute intestinal obstruction, 342  
amoebic, 334  
complications of, 333  
course of, 332  
etiology of, 330  
morbid anatomy of, 330  
prognosis of, 333  
prophylaxis of, 333  
treatment of, 333
- Dyspepsia, acute gastric, 309  
course of, 310  
symptoms of, 309  
treatment of, 310
- Dyspnoea, ascites and abdominal tumors  
as a cause of, 362  
in atelectasis, 93  
forms of, 361  
cardiac, 361  
laryngeal, 361  
pulmonary, 361  
in heart disease, 453
- Dysuria, 616  
in measles, 153
- E**AR, syphilis of, 265
- Echolalia, 498
- Eczema, etiology of, 641  
squamous, treatment of, 643  
treatment of, 642  
varieties of, 639, 640
- Emphysema in bronchiectasis, 373  
of the lungs, 366  
frequency of, 366  
morbid anatomy of, 366  
physical signs of, 367, 369  
prognosis of, 370  
symptoms of, 367  
treatment of, 370  
in pertussis, 203
- Empyema, 410  
bacteriology of, 412  
bilateral, 430  
deformity after excision of rib, 428  
diagnosis of, 416  
displacement of viscera in, 420  
of pleural fold in, 419  
etiology of, 411  
frequency of, 410  
hemorrhagic, 431  
morbid anatomy of, 411  
perforating, 423  
course of, 424
- Empyema, perforating, prognosis of, 424  
treatment of, 425  
physical signs of, 416, 422  
symptoms of, 415  
treatment of, 426  
of adhesions, 429  
excision of rib, 429  
incision of, 426  
sinus in, 429  
unexpanded lung in, 429
- Enanthema in scarlet fever, 133
- Endocarditis, acute, bacteriology, 442  
course of, 449  
etiology of, 442  
morbid anatomy of, 443  
physical signs of, 447  
prognosis of, 449  
symptoms of, 444  
treatment of, 449  
in cerebrospinal meningitis, 194  
and follicular amygdalitis 301  
healed, 451  
in scarlet fever, 131  
septic or malignant ulcerative, 445  
diagnosis of, 447  
symptoms of, 446
- Endopericarditis in sepsis, 97
- Enema, in constipation, 44  
stimulating, 44
- Enemata, in acute intestinal obstruction, 344  
in constipation, 338  
rectal, 42
- Enteric catarrh. *See* Enterocolitis.
- Enteritis follicularis. *See* Dysentery.
- Enteroclysis, 42
- Enterocolitis. *See* Dysentery.  
acute and subacute, 328  
etiology of, 328  
morbid anatomy of, 328  
symptoms of, 329  
treatment of, 329
- Enuresis, diagnosis of, 633  
diurna, 632  
nocturna, 632  
symptoms of, 633  
treatment of, 633
- Epiglottitis, method of hooking up, in intubation, 231
- Epilepsy, convulsions in, 487  
diagnosis of, 488  
etiology of, 487  
symptoms of, 487  
treatment of, 488
- Epiphyses, osteomyelitis of, 597
- Epstein's pearls, 96
- Erb's palsy, 113  
etiology of, 518  
prognosis of, 518  
symptoms of, 518  
treatment of, 518
- Erosions, dental, 279
- Erythema exudativum, 645



- Erythema multiforme, 645  
     treatment of, 645  
     nodosum, 551, 645  
     cardiac lesion in, 645
- Erythrocytes, 570
- Estlander's operation, 430
- Exanthema in diphtheria, 219
- Exanthemata, 118  
     frequency of, 18  
     incubation in, 118
- Extubation, 233
- Eye symptoms, in cerebrospinal meningitis, 192  
     in late hereditary syphilis, 263
- Eyes, nitrate of silver instillation, Credé method, 44
- F**ACE in parotitis, 20  
     symmetry of, in adenitis, 20
- Facial palsies in tuberculous meningitis, 256  
     paralysis. *See* Paralysis, facial.  
         during birth, 113  
         expression in, 21  
         in mastoid disease, 604  
         in otitis, 600
- Fatty degeneration, acute, of newborn infant, 105  
     diagnosis of, 106  
     etiology of, 106  
     morbid anatomy of, 106  
     prognosis of, 106  
     symptoms of, 106  
     treatment of, 106
- Feeding after the sixth month, 81  
     amount of daily albumin, fat, and carbohydrates, 83  
     artificial, 64, 80  
     eighteenth to twenty-fourth month, 82  
     infants and children, 48  
     ninth to twelfth month, 81  
     quantity of food to be given, 73  
     sick infants and children, 83  
     twelfth to eighteenth month, 82
- Filth infections, frequency of, 18
- Folliculitis abscedens, 646
- Fontanelles, closure of, 536  
     condition of, 20  
     in tuberculous meningitis, 254
- Food, infant, composition, 74
- Foot-and-mouth disease of cattle, and aphthous stomatitis, 282
- Foramen ovale, open, 441
- Fremitus, 358  
     method of ascertaining, 24
- Friedreich's disease, 519
- Fungus, umbilical, 99  
     treatment of, 99
- Furunculosis, 646
- G**AIT, ataxic, 31  
     limping, 32
- Gangrene of lung, in bronchiectasis, 373  
     in bronchopneumonia, 399
- Gastric juice, ferments of, 307  
     free HCl in, 306  
     combined HCl in, 306  
     spasm, congenital, 313
- Gastro-enteritis, acute, 316  
     classification of, 316  
     course and prognosis of, 317  
     diagnosis of, 321  
     in diphtheria, 215  
         treatment of, 234  
     morbid anatomy of, 317  
     nephritis in, 317, 319  
     otitis in, 598  
     in pertussis, 204  
     prophylaxis of, 321  
     role of bacteria in, 317  
     symptoms of, 318  
     treatment of, 321
- Girls, loss of weight in newborn, 53  
     weight and height, 57
- Glandular apparatus in hereditary syphilis, 268
- Glandular fever, 189  
     diagnosis of, 190  
     duration of, 190  
     etiology of, 189  
     symptoms of, 189  
     treatment of, 190
- Glottis, spasm of, 484
- Gonorrhoeal urethritis, 636  
     ophthalmia in newly born infant, 44
- Gruels, dextrinized, 78
- Gums, incision of, 281
- Gyrospasm, 498
- H**ABIT movements, 498  
     Haematoma of sternomastoid muscle, 115
- Hæmaturia, 617  
     in carcinoma of kidney, 629  
     causes of, 617
- Hæmoglobin, 571  
     in fœtus, 571  
     in infancy, 572  
     in newly born infant, 572
- Hæmoglobinuria, causes of, 618  
     epidemic in newly born infant, 107  
     paroxysmal, 618  
     treatment of, 618
- Hæmophilia, 583  
     etiology of, 583  
     nature of, 583  
     symptoms of, 563  
     treatment of, 583
- Hands of nurse, care of, 46
- Head, bones of, injury to, 596  
     syphilis of, 596  
     examination of, 20  
     measurements, 56  
     position in Pott's disease, 20  
     shape of, 20

- Head-nodding, 498  
Hearing, 48  
Heart, 433  
    apex-beat, 433  
    auscultation, 434  
    chronic disease of, 450  
        etiology of, 450  
        frequency of, 450  
        physical signs of, 450  
        prognosis of, 453  
        symptoms of, 453  
        treatment of, 454  
    congenital disease of, 437  
    dilatation of, 466  
        in pertussis, 203  
        symptoms of, 466  
        treatment of, 466  
    disease in bronchiectasis, 373  
    displacement in pleurisy, 421  
    hypertrophy of, 466  
    irritable, 451  
    paralysis of, in diphtheria, 216  
    percussion of, 434  
    physical examination of, 433  
    position of, 433  
    size of, 433  
    sounds, character of, 434  
Hemi-anesthesia in pertussis, 204  
Hemiplegia in cerebrospinal meningitis, 194  
    in diphtheria, 218  
    in pertussis, 204  
    spastic. *See* Cerebral palsy.  
Hemorrhage, idiopathic, from umbilicus, 102  
    etiology of, 102  
    occurrence of, 102  
    symptoms of, 102  
    treatment of, 103  
    in pertussis, 204  
Hemorrhagic diathesis, 582  
Hernia, umbilical, 103  
    symptoms of, 103  
    treatment of, 103  
Herpes labialis in cerebrospinal meningitis, 192  
    of tonsil, 221, 302  
Heubner-Hoffmann mixture, 67  
Hippus in tuberculous meningitis, 255  
Hutchinson's teeth, 278  
Hydremia, 573, 616  
Hydrancephaloid in cholera infantum, 320  
    diagnosis of, 502  
    facial expression in, 21  
Hydrocephalus, acute, 500  
    course of, 500  
    symptoms of, 500  
    chronic external, diagnosis of, 502  
        symptoms of, 503  
    internal, 500  
        diagnosis of, 502  
        etiology of, 500  
Hydrocephalus, chronic, internal, morbid  
    anatomy of, 500  
    prognosis of, 502  
    symptoms of, 501  
    treatment of, 503  
    facial expression in, 21  
Hydromyelocele, 529  
Hydronephrosis, 627  
Hydrorhachis, 528  
Hydrotherapy, 34  
    in bronchopneumonia, 405  
    in lobar pneumonia, 389  
    in measles, 157  
    in scarlet fever, 138  
    in typhoid fever, 178  
Hygiene of infancy and childhood, 44  
Hypertrophic pyloric stenosis. *See* Pyloric stenosis, 313  
Hypodermoclysis, 36  
    in acute gastro-enteritis, 323  
Hysteria, 476  
    diagnosis of, 478  
    disturbances of sensation, 478  
    duration of, 479  
    etiology of, 476  
    motor manifestations in, 477  
    psychic or mental, 476  
    symptoms of, 476  
    treatment of, 479  
**I**CHTHYOSIS, congenital, 648  
    symptoms of, 648  
    treatment of, 648  
Icterus, catarrhal, 609  
    infectious, 609  
    neonatorum, 111  
        etiology of, 111  
        symptoms of, 111  
        treatment of, 111  
    in the newly born, 110  
Idiocy, amaurotic, 504  
    diagnosis of, 505  
    etiology of, 504  
    morbid anatomy of, 504  
    ocular changes in, 505  
    spastic symptoms of, 505  
    symptoms of, 504  
    with dwarfism, 565  
    Mongolian, 565  
Ileocolitis. *See* Dysentery.  
Ileotyphus. *See* Typhoid fever.  
Impetigo contagiosa, 640  
Incubators, 85  
    indication for use of, 87  
Infancy, definition of, 17  
Infant foods, 77  
    mode of holding, for examination, 23  
Infantalism, 565  
    dental, 280  
Infantile paralysis, gait in, 32  
Infants, premature, 85  
Infarction, uric acid, 619  
Infectious diseases, acute, frequency of, 18



Influenza and angina, 186  
 and bronchitis, 186  
 complications of, 187, 189  
 and diarrhoea, 186  
 etiology of, 185  
 incubation in, 185  
 morbid anatomy of, 185  
 and meningitis, 187  
 and nephritis, 188  
 and otitis, 188, 598  
 and pneumonia, 187  
 symptoms of, 185  
 Infarctures in scorbutus, 592  
 Intertrigo, 641  
     treatment, 644  
 Intestinal obstruction, acute, 339  
     diagnosis of, 342  
     enemata in, 343  
     frequency of, 340  
     hemorrhage in, 341  
     prognosis of, 342  
     spontaneous cure of, 343  
     symptoms of, 340  
     tenesmus in, 341  
     treatment of, 343  
     tumor in, 341  
     vomiting in, 340  
 Intestine, bacteria of, 308  
     ferments of, 307  
 Intramuscular abscess in cerebrospinal  
 meningitis, 192  
 Intubation, 227  
     built-up tubes, 234  
     dangers of, 233  
     feeding after, 234  
 Intussusception, 339. *See* Obstruction,  
 acute intestinal.  
 Invertin, 308  
 Irrigation, rectal, 42  
 Irritable heart, 451  
  
**JAMES'** bottles, 429  
 Jaundice, 19, 609  
     bacteriology of, 610  
     catarrhal, 609  
     duration of, 611  
     morbid anatomy of, 610  
     occurrence of, 610  
     symptoms of, 610  
     treatment of, 611  
 Joints, affections of, in varicella, 161  
     crepitus, 28  
     examination of, 27  
     hemorrhage into, 583  
     in measles, 153  
     mobility, 28  
     osteomyelitis of, 597  
     in septic infection, 96  
  
**KERATITIS**, in cerebrospinal mening-  
 itis, 192  
 Kernig's symptom, 31, 192  
     in tuberculous meningitis, 257

Kidney, carcinoma, 628  
     diagnosis of, 630  
     duration of, 630  
     hæmaturia in, 629  
     symptoms of, 629  
     urine in, 629  
     cysts of, 627  
     diseases of, 614  
     displaced, 614  
     floating, 27, 614  
     growths of, treatment of, 631  
     new growths of, 627  
     palpation of, 614  
     sarcoma of, 628  
         urine in, 628  
     in scarlet fever, 130  
     tuberculosis of, 630  
     weight, 614  
 Koomyass, 80  
 Kyphosis, normal, 28  
  
**L** ABIA, cellular atresia of, 617  
     Laborde's method in asphyxia, 91  
 Lactase, 308  
 Laryngeal disease in bronchiectasis, 373  
 Laryngismus stridulus, 484  
     complications in, 486  
     diagnosis of, 486  
     etiology of, 484  
     lymphatism with, 581  
     morbid anatomy of, 485  
     occurrence of, 484  
     prognosis of, 486  
     symptomatology of, 485  
     and the thymus, 568  
     treatment of, 486  
 Laryngitis, acute catarrhal, 303  
     etiology of, 303  
     symptoms of, 303  
     treatment of, 304  
 Laryngospasm and tetany, 482  
 Larynx, infantile, 232  
     tuberculosis of, 251  
 Leucocytes, number in a cubic milli-  
 metre, 571  
 Leucocythæmia, 577  
 Leucocytosis, digestive, 571  
     in diphtheria, 571  
     inflammatory, 571  
     in lobar pneumonia, 383  
     in measles, 571  
     in pertussis, 203  
     in post-hemorrhagic anæmia, 571  
     in pneumonia, 571  
     in pseudoleukæmic anæmia, 577  
     in purpura hemorrhagica, 584  
     in rheumatism, 571  
     in scarlet fever, 132, 571  
     in sepsis, 571  
 Leucopenia, in cachexia, 571  
     in malaria, 571  
     in tuberculosis, 571  
     in typhoid fever, 571

- Leukemia, 577  
   acute, symptoms of, 579  
   blood-changes in, 578  
   chronic, symptoms of, 580  
   etiology of, 578  
   forms of, 578  
   lymphatic, 578  
   myelogenous, 578  
   occurrence of, 578  
 Lewi fat analysis, 62  
 Lichen scrofulosorum, 238  
 Liebig's food, 322  
   mixtures, 79  
 Lingua geographica, 291  
 Lithiasis, 19  
   and dysuria, 616  
 Liver abscess, 612  
   in bronchiectasis, 373  
   acute yellow atrophy of, 613  
   cirrhosis of, 611  
   diseases of, 607  
   displacement of, by empyema, 609  
   in pleurisy, 420  
   enlargement, diagnosis from kidney  
   tumors, 609  
   enlargement of, 609  
   examination of, 607  
   fatty degeneration of, 612  
   and varicella, 161  
   ferments, 307  
   palpation of, 607  
   percussion of, 607  
   phantom tumor of, 608  
   in rachitis, 609  
   in sepsis, 97  
   superficial dulness of, 608  
   syphilis of, 612  
     hereditary, 272  
     late, 265  
 Lobar pneumonia mistaken for appendi-  
   citis, 347  
 Lordosis, normal, 28  
 Lumbar puncture, 468  
   abnormal cerebrospinal fluid,  
     468  
   in cerebrospinal meningitis, 195  
   gross appearances of cerebro-  
   spinal fluid, 468  
   indications for, 471  
   instrument for, 471  
   method of, 470  
   normal cerebrospinal fluid, 468  
   operation of, 470  
   place of puncture, 470  
   specific gravity of cerebrospinal  
   fluid, 468  
   in tuberculous meningitis, 260  
 Lung, atelectasis of, 93  
   gangrene in scarlet fever, 132  
 Lungs, emphysema of, 366  
   in hereditary syphilis, 267  
   normal limits of, 358  
 Lymphadenitis, chronic, 558  
   Lymphadenitis, chronic, symptoms of, 558  
     treatment of, 559  
     retropharyngeal. *See* Retropharyn-  
       geal abscess, 294  
 Lymphatism, 581  
 Lymph-nodes, enlargements of, 556  
   in glandular fever, 190  
   in measles, 153  
   of retropharynx, 295  
   in scarlet fever, 134  
  
**M**ACEWEN'S sign in cerebrospinal  
   meningitis, 194  
   in tuberculous meningitis, 260  
 Macroglossia, 291  
 Malaria, anaemia in, 572  
 Malarial fever, 179  
   blood in, 180  
   diagnosis of, 182  
   etiology of, 180  
   morbid anatomy of, 183  
   prognosis of, 182  
   repeated attacks of, 182  
   symptoms of, 181  
   treatment of, 183  
 Malted foods, 78  
 Mania, in scarlet fever, 130  
 Marasmus. *See* Atrophy.  
 Mastitis in newborn infants, 48  
 Mastoid disease, enlarged lymph-nodes  
   in, 603  
   facial paralyses in, 604  
   pain in, 603  
   in scarlet fever, 603  
   temperature in, 603  
   tumefaction in, 604  
   inflammation of, course of, 605  
   diagnosis of, 604  
   etiology of, 602  
   frequency of, 602  
   in measles, 154  
   otoscopic examination in, 603  
   physical signs of, 603  
   prophylaxis, 605  
   symptoms of, 602  
   treatment of, 605  
   region, general, 601  
 McBurney's point, 347  
 Measles, 143  
   amaurosis in, 153  
   and antitoxin eruption, 156  
   blood conditions in, 153  
   bronchitis, bronchopneumonia, and  
   atelectasis in, 150  
   bronchopneumonia in 398  
   conjunctivitis in, 153  
   definition of, 143  
   diagnosis of, 154  
   diarrhoea in, 152  
   diphtheria in, 150, 154, 219  
     treatment of, 158  
   and drug eruptions, 156  
   dysuria in, 153



- Measles, enanthema of, 147  
 endocarditis in, 152  
 exanthema of, 148  
   and syphilis, 156  
 heart in, 152  
 hemorrhagic, 149  
 immunity in, 144  
 incubation in, 144  
 intestinal complications in, 152  
 joints in, 153  
 leucocytosis in, 571  
 mastoid inflammation in, 154  
 meningitis in, 153  
 myocarditis in, 152  
 nephritic complications and sequelae of, 152  
 nephritis in, 152  
 nose, pharynx, and larynx in, 149  
 osteomyelitis in, 153  
 otitis in, 154  
 photophobia in, 153  
   and pertussis, 153  
 pneumonia in, 151  
 prognosis of, 154  
 prophylaxis of, 156  
   and Rötheln, 155  
   and scarlet fever, 155  
 sequelae of, 154  
 stomatitis in, 153  
 symptoms of, 145  
 temperature in, 146  
 treatment of complications, 157  
   general, 156  
   tuberculosis in, 152  
 Meconium, 53  
 Meigs' mixture, 66  
 Melena neonatorum, 104  
   diagnosis of, 105  
   morbid anatomy of, 104  
   prognosis of, 105  
   symptoms of, 105  
   treatment of, 105  
   spurious, 105  
 Melancholia in pertussis, 204  
   in scarlet fever, 131  
 Meningitis, cerebrospinal. *See* Cerebro-spinal meningitis.  
   influenza and, 187  
   in measles, 153  
   in pneumonia, 399  
     diagnosis of, 260  
     posterior basic, 195  
     staphylococcic, 195  
     streptococcic, 195  
     treatment of, 261  
   tuberculous, 252  
     Chvostek symptom in, 255  
     diagnosis of, 259  
     duration of, 261  
     etiology of, 252  
     Kernig's symptom in, 255  
     lumbar puncture in, 260  
     morbid anatomy of, 252  
   Meningitis, tuberculous, occurrence of, 252  
     ordinary type of, 254  
     prognosis of, 261  
     rapid course of, 258  
     symptoms of, 254  
     temperature in, 256  
     Trousseau symptom in, 255  
 Meningo-encephalocele, 528  
 Meningocele spinalis, 529  
 Menstruation, effect on breast-milk, 60  
 Microdontism, 280  
 Miliaria alba, 647  
   rubra, 647  
 Milk, acidity of, 70  
   in breasts of infants, 48  
   in breasts of nurse, 50  
   human, analysis of fats, 61  
     composition of, 58  
     excretion of drugs in, 59  
     microscopical examination of, 64  
     proteids in, 58  
     reaction of, 59  
     specific gravity of, 59  
     variations in daily composition of, 59  
   modifications of, home, 75  
     laboratory, 75  
   mother's, insufficient, 51  
   raw, in infant feeding, 72  
 Mongolian idiocy, palpebral fissure in, 21  
 Monoplegia in pertussis, 204  
 Morbidity of acute infectious diseases, 18  
   in childhood, 18  
   diseases of the kidney, 18  
   exanthemata, 18  
   gastro-enteric diseases, 18  
   of newborn, 17  
   respiratory diseases, 18  
 Morbus maculosus Werlhofii, 584  
 Mouth, bacteria, 308  
   care of, 46  
   in breast-fed infants, 286  
   diseases of, 277  
   infant, in sleep, 20  
   normal, characteristics of, 277  
   reaction, secretions of, 305  
   toilet, in bottle-fed infants, 286  
   washing of, 45  
 Mucous colitis. *See* Mucous disease.  
   disease, 335  
     symptoms of, 336  
     treatment of, 338  
   membranes in late syphilis, 265  
 Mumps, 196  
   complications of, 199  
   course of, 199  
   diagnosis of, 200  
   etiology of, 196  
   morbid anatomy of, 197  
   prognosis of, 200  
   submaxillary involvement in, 198  
   symptoms of, 197

- Mumps, treatment of, 200
- Murmurs, arterial, 456
- cardiac, accidental, 455
- characters of, 455
- in congenital heart disease, 437
- venous, 456
- Muscle, atrophy of, 30
- hypertrophy of, 30
- reflex, 30
- Myelocystocele, 529
- Myelomeningocele, 529
- Myocarditis, diagnosis of, 458
- etiology of, 457
- morbid anatomy of, 456
- in scarlet fever, 131
- treatment of, 458
- Myotonia, 483
- N**EPHRITIS, acute, 619
- diffuse, 619
- exudative, 619
- parenchymatous, 619
- treatment of, 623
- anasarca in, 622
- in cholera infantum, 320
- chronic diffuse, 624
- symptoms of, 624
- in diphtheria, 215
- influenza and, 188
- in measles, 152
- in pertussis, 203
- in scarlet fever, 130
- uremia in, 622
- in varicella, 161
- Nerves, morbid anatomy of, in diphtheria, 210
- Neuritis, multiple, 515
- course of, 516
- diagnosis of, 517
- etiology of, 515
- morbid anatomy of, 515
- sensory disturbances in, 516
- symptoms of, 516
- treatment of, 518
- Newborn infant, asphyxia of, 89
- definition of, 17
- osteomyelitis in, 597
- placing at the breast, 52
- polycythemia in, 570
- Nipples, bottle, care of, 46
- fissures of, 52
- inflamed, 52
- Nodes, mesenteric, enlarged, 26
- Noma, occurrence of, 291
- symptoms of, 290
- treatment of, 291
- Normoblasts, 573
- Nose, intra-uterine deformity of, in hereditary syphilis, 269
- syringing of, 37
- Nursery, temperature of, 45
- Nursing bottle, form of, 72
- at breast, intervals of, 52
- Nursing inefficient, signs of, 53
- infant at breast, contraindications to nursing, 51
- intervals of, 53
- period, morbidity of, 18
- Nursings, number of daily, 74
- number of, in twenty-four hours, 53
- Nutans spasmus, 498
- Nutation of head, nystagmus in, 22
- Nystagmus, 22, 498
- in albinism, 22
- in amaurotic idiocy, 22
- in rachitic infants, 22
- O**BSTETRICAL palsy, 518
- Ocular symptoms in otitis, 260
- Oculomotor paralysis in pertussis, 204
- Oesophageal stricture due to ingestion of alkali, 287
- Omphalitis, 99
- Omphalorrhagia, 101
- Ophthalmia, diphtheritic, 218
- neonatorum, 109
- prognosis of, 110
- treatment of, 110
- Opisthotonos in tetanus, 108
- in tuberculous meningitis, 255
- Optic neuritis in cerebrospinal meningitis, 192
- Orbital muscles, paralysis of, in cerebrospinal meningitis, 192
- Osteomyelitis, 596
- diagnosis of, 597
- from scorbutus, 597
- from syphilis, 597
- etiology of, 596
- of joints, 597
- in measles, 153
- in newborn infant, 597
- prognosis of, 598
- in scarlet fever, 129, 597
- in septic infection, 96
- symptoms of, 597
- treatment of, 598
- tuberculous, 598
- Otitis, 598
- acute suppurative, 599
- bacteriology of, 598
- in bronchopneumonia, 399
- in cerebrospinal meningitis, 598
- complications of, 601
- course of, 600
- diagnosis of, 601
- in diphtheria, 598
- etiology of, 598
- frequency of, 598
- in gastro-enteritis, 598
- in influenza, 188, 598
- in measles, 154
- media catarrhalis, 599
- purulent, temperature, 600
- and meningitis, diagnosis of, 260
- morbid anatomy of, 599



Otitis, and ocular symptoms, 260  
 in pertussis, 598  
     prognosis of, 601  
 in scarlet fever, 127, 133, 135  
     symptoms of, 599  
 in typhoid fever, 598  
 in varicella, 161  
 Oxyuris vermicularis, 354

**P**ACK, cold, 35  
 Palpitation in chronic heart disease, 451  
 Palsies, acute, 509  
     birth, 114, 509, 510  
     prenatal, 509, 510  
 Palsy, cerebral, acute, 510  
     due to otitis, 514  
     facial, 512  
         nuclear, facial expression in, 21  
         post-natal, 509  
         rheumatic, 513  
         treatment, 515  
 Pancreas in congenital syphilis, 268  
     physiological facts, 308  
 Paralysis, acute atrophic, atrophy of muscle, 522  
     course of, 522  
     diagnosis of, 523  
     etiology of, 520  
     morbid anatomy of, 523  
     occurrence of, 520  
     onset of, 521  
     paralysis in, 521  
     prognosis of, 522  
     sequelæ of, 522  
     symptoms of, 521  
     treatment of, 524  
   in diphtheria, 217  
     treatment of, 234  
   essential, 520  
   infantile, 520  
   pseudo-hypertrophic muscular, 525  
     complications of, 527  
     course of, 526  
     diagnosis of, 527  
     electrical reaction of muscle in, 526  
     morbid anatomy of, 527  
     occurrence and etiology of, 525  
     prognosis of, 527  
     reflexes in, 526  
     symptoms of, 525  
     treatment of, 527  
     varieties of, 527  
   of soft palate in diphtheria, 221  
 Paraneuphritis, 632  
     symptoms of, 631  
     treatment of, 632  
 Paraplegia. *See* Cerebral palsy.  
     spastic, 32  
 Parasites, intestinal, 352  
     diagnosis of, 352

Parotitis, epidemic. *See* Mumps.  
 Pasteurization, 70  
 Pasteurized milk in scorbutus, 589  
 Patellar reflex, 30  
 Pavor nocturnus, 488  
     prognosis of, 489  
     symptoms of, 489  
     treatment of, 489  
 Pemphigus neonatorum, 649  
     nature and symptoms of, 649  
     prognosis of, 650  
     treatment of, 650  
     in septic infection, 96  
 Peptonized milk, 80  
 Percussion of the chest, 25  
 Pericardial effusion, amount and diagnosis of, 461  
     and pleuritic effusion, differential diagnosis, 464  
 Pericarditis, diagnosis of, 463  
     etiology of, 458  
     form of dullness of the effusion, 462  
     forms of, 459  
     heart-apex in, 461  
     in measles, 152  
     morbid anatomy of, 459  
     occurrence of, 458  
     physical signs of, 460  
     in pneumonia, 399  
     in scarlet fever, 132  
     symptoms of, 460  
     treatment of, 464  
 Pericardium, adherent, 465  
     symptoms, 465  
     tuberculosis of, 252  
 Perifolliculitis abscedens, 646  
 Perinephritic abscess and appendicitis, 347  
 Perinephritis, 632  
 Periosteum, hemorrhages into, in scorbutus, 590  
 Periostitis, hemorrhagic. *See* Scorbutus.  
 Peritoneum, tuberculosis of. *See* Tuberculosis of peritoneum.  
 Peritonitis and acute intestinal obstruction, 342  
     simple chronic, 251  
     in vulvovaginitis, 636  
 Pertussis, antipyrin in treatment of, 205  
     aphasia in, 204  
     blindness in, 204  
     blood in, 203  
     and bronchitis, 202  
     bronchopneumonia in, 397  
     cardiac dilatation in, 203  
     catarrhal stage of, 201  
     cocaine in, treatment of, 205  
     complications of, 203  
     convulsions in, 204  
     definition of, 200  
     diagnosis of, 204  
     in diphtheria, 219  
     emaciation in, 204  
     emphysema in, 203

- Pertussis, etiology of, 201  
     frequency of, 18  
     gastro-enteritis in, 204  
     hallucinations in, 204  
     hemianæsthesia in, 204  
     hemiplegia in, 204  
     hemorrhages in, 204  
     in measles, 153  
     melancholia in, 204  
     monoplegia in, 204  
     morbid anatomy of, 201  
     mortality and prognosis of, 204  
     nephritis in, 203  
     in the newly born infant, 200  
     occurrence of, 200  
     oculomotor paralysis in, 204  
     ozone in, 205  
     spasmodic stage of, 202  
     symptoms of, 201  
     treatment of, bromoform in, 205  
         medicinal, 205  
         prophylaxis, 204  
     urine in, 203  
 Peliosis rheumatica, 551, 585  
 Phalanges, deformities of, in rachitis, 539  
 Phlebitis, umbilical, 101  
     treatment of, 101  
 Photophobia, 22  
     in measles, 153  
 Pin worm, 354  
 Pleura, tuberculosis of, 252  
 Pleural fold displacement of, in pleurisy  
     with effusion, 419  
     and pericardial effusions, diagnosis of, 464  
 Pleurisy, dry, forms of, 409  
     diagnosis of, 410  
     etiology of, 409  
     prognosis of, 410  
     symptoms of, 409  
     treatment of, 410  
     heart displacement in, 421  
     hemorrhagic, 430  
     in measles, 152  
     with effusion. *See* Empyema.  
 Pleuritis in diphtheria, 215  
     in scarlet fever, 132  
 Pleuropericardial friction, 463  
 Pleuroplegia, 21  
 Pneumonia, broncho-. *See* Bronchopneumonia.  
     influenza and, 187  
     lobar, crisis of, 378  
         bacteriology of, 377  
         the blood in, 383  
         bradycardia, 382  
         cerebral symptoms in, 382  
         cough in, 382  
         diagnosis of, 388  
         dyspnœa in, 382  
         etiology of, 377  
         hydrotherapy in, 389  
         lysis in, 378  
     lobar, morbid anatomy of, 376  
         nervous symptoms of, 382  
         occurrence of, 375  
         physical signs of, 383  
         prognosis of, 387  
         pseudocrisis in, 378  
         remittent temperature, 380  
         seat of disease in, 376  
         short course of, 385  
         subnormal temperature of, 380  
         symptoms of, 377  
         temperature in, 379  
         treatment of, 389  
     meningitis in, 399  
     osteomyelitis in, 597  
     otitis in, 598  
     in scarlet fever, 132  
     and typhoid fever, 388  
     in varicella, 161  
 Pneumothorax in pertussis, 204  
 Poikilocytosis in anæmia, 573  
 Poliomyelitis, anterior, acute, 520  
 Polypi, intestinal, 26  
 Polyuria, 553  
 Posterior basic meningitis, 195  
 Pott's disease, 29  
     spinal deformity, 538  
 Premature infants, 85  
     causes of death, 88  
     management of, 85  
     morbid anatomy of, 89  
     prognosis of, 88  
     rectal temperature of, 85  
 Proctitis, 351  
     treatment of, 351  
 Progressive muscular atrophy, juvenile form, 521  
 Pseudoleukæmic anæmia, 575  
 Pseudoparalysis in rachitis, 542  
 Psoas spasm, 30  
 Psychical derangements in diphtheria, 218  
 Psychoses in pertussis, 204  
 Pulmonary artery, stenosis of, 439  
     murmur in, 440  
     physical signs of, 439  
 Pulmonary resonance, 358  
 Puncture, exploratory, of the chest, 422  
 Purpura hæmorrhagica, 584  
     course of, 584  
     diagnosis of, 585  
     etiology of, 584  
     treatment of, 585  
     Henoch's, 586  
     rheumatica, 585  
         etiology of, 585  
         prognosis of, 585  
         symptoms of, 585  
         treatment of, 585  
     simple, 582  
         etiology of, 582  
         prognosis of, 582  
         symptoms of, 582



- Purpura, simple, treatment of, 583  
 Pyelitis with cystitis, 637  
 Pylorus, congenital stenosis of, 313  
   hypertrophic stenosis of, 313  
     etiology of, 314  
     morbid anatomy of, 314  
     prognosis of, 315  
     symptoms of, 314  
     treatment of, 315
- R**ACHISCHISIS totalis, 527  
 Rachitis, acute. *See* Scorbutus.  
 Rachitis, anaemia in, 540  
   blood in, 540, 542  
   of bones, 595  
   cardiac area in, 24  
   and dactylitis, 539  
   deformities of extremities, 540  
   diagnosis of, 541  
   duration of, 541  
   etiology of, 533  
   expression of face in, 21  
   fontanelles in, 536  
   head in, 535  
   hemorrhagic. *See* Scorbutus.  
   hydrocephalus in, 541  
   intestinal disturbances in, 540  
   liver in, 540, 609  
   morbid anatomy of, 534  
   morbidity of, 18  
   nervous system in, 541  
   occurrence of, 541  
   pain in, 537  
   pelvic deformities of, 539  
   phosphorus in the treatment of, 544  
   prognosis of, 542  
   relation to scorbutus, 589  
   spine in, 538  
   spleen in, 540, 569  
   symptoms of, 535  
   tarda, 542  
   treatment of, 543  
 Rectum, anatomy of, 349  
   exploration of, 27  
   polypus of, 351  
     diagnosis of, 352  
     prognosis of, 352  
     treatment of, 352  
 Red blood-cells, characters of, 570  
 Respirations, normal number of, 357  
   in rest and unrest, 20  
 Respiratory apnoea in retropharyngeal  
   abscess, 297  
   apparatus, frequency of diseases of,  
   18  
   tract, diseases of, 357  
 Retropharyngeal abscess, acute, 294  
   course of, 296  
   diagnosis of, 296  
   etiology of, 295  
   forms of, 295  
   frequency of, 295  
   prognosis of, 297  
 Retropharyngeal abscess, acute, symptoms  
   of, 296  
   treatment of, 297  
   characteristic breathing in, 20  
   in diphtheria, 216  
   in scarlet fever, 129  
 Revaccination, 166  
 Rheumatic fever, 547  
 Rheumatism, acute articular, chorea in,  
   549  
   endocarditis in, 549  
   nature of, 547  
   occurrence of, 548  
   prognosis of, 550  
   symptoms and types of, 548  
   treatment of, 550  
   and chorea, 490, 549  
   gonorrhoeal, 551  
   leucocytosis in, 571  
   muscular, 551  
 Rheumatoid affections, 551  
   arthritis, 545  
     goitre in, 547  
     occurrence of, 545  
     prognosis of, 547  
     symptoms of, 546  
     treatment of, 547  
 Rickets, foetal, 565  
   scurvy. *See* Scorbutus.  
 Ringworm of the tongue, 291  
 Roger, maladie de, 441  
 Rosary of the ribs in rachitis, 536  
 Rötheln, 140  
   complications of, 143  
   desquamation in, 142  
   diagnosis of, 143  
   enanthera of, 142  
   exanthema of, 141  
   eruption on the genitals in, 143  
   lymph-nodes in, 143  
   measles and, 155  
   occurrence of, 141  
   prodromal period of, 141  
   spleen in, 143  
   temperature curve of, 142  
   treatment of, 143  
 Round worms, 353
- S**ALT solution, normal, 36  
 Sarcoma of kidney, 628  
   diagnosis of, 628  
   symptoms of, 628  
 Scalp, abscess of, 117  
   phlegmon of, 117  
 Scarlet fever, abscesses in the skin, 125  
   acute dilatation of heart in, 132  
   amaurosis in, 130  
   anaemia in, 132  
   the angina of, 122  
   antipyretics in, 138  
   antitoxin in, 139  
   aphasia in, 131  
   bacteriology of, 135

- Scarlet fever, blood in, 132  
 bronchopneumonia in, 398  
 buccal mucous membrane in, 123  
 chorea in, 133  
 cystitis in, 637  
 the desquamation of, 126  
 diagnosis of, 133  
 diarrhoea in, 132  
 diphtheria in, 123  
 diphtheroid in, 123  
 drug eruptions in, diagnosis of, 134  
 the ear, 127  
 endocarditis in, 131  
 etiology of, 118  
 evanescent forms of, 120  
 the exanthema or rash, 124  
 the eye, 128  
 the fever, 125  
 frequency of, 18  
 general course of, 121  
 immunity, 119  
 incubation of, 119  
 joints in, 129  
 kidneys in, 130  
 leucocytosis in, 571  
 lungs in, 132  
   complications, treatment of, 140  
 the lymph-nodes in, 128  
 malignant cases of, 122  
 mania in, 130  
 mastoid disease in, 603  
 measles and, 155  
 melancholia in, 131  
 membranous angina, 123  
 mode of infection, 119  
 the mouth in, 129  
 myocarditis in, 131  
 necrosis of the nasal cartilages, 124  
 nephritis in, 130, 622  
 nervous diseases in, 133  
 the nose and nasal passages in, 124  
 occurrence of, 119  
 osteomyelitis in, 597  
 otitis, treatment of, 140  
 periarticular abscess in, 130  
 pleura in, 132  
 prognosis of, 134  
 prophylaxis of, 137  
 rash, pruritus with, 125  
 retropharyngeal abscess in, 129  
 sequelae of, 132  
 stomach and intestines in, 132  
 symptoms of, 120  
 the tongue in, 124  
 treatment of, 136, 138  
   of heart, 138  
   of joints, 140  
   of lymph-nodes, 139  
   of nephritis, 139  
   of throat and nose, 139
- Scarlet fever, tuberculosis in, 133  
   uremia in, 131  
   vomiting in, 132  
 Scarlatinal diphtheroid, 222  
   nephritis, frequency of, 18  
   rheumatism, 129  
   prognosis of, 135  
 Sclerema adiposum, 112  
   diagnosis of, 112  
   etiology of, 113  
   in cholera infantum, 320  
   neonatorum, 112  
 Sclerodactylia, 112  
 Sclerodema, 112  
 Scorbutus, bone changes in, 590  
   diagnosed from osteomyelitis, 597  
   from purpura, 586  
   from syphilis, 593  
   disability in, 591  
   fracture and infraction, 592  
   infantile, 588  
     diagnosis of, 593  
     duration of, 592  
     etiology of, 589  
     history of, 588  
     mild cases of, 590  
     morbid anatomy of, 590  
     nature of the affection, 588  
     occurrence of, 588  
     prognosis of, 592  
     severe cases, 591  
     symptoms of, 590  
     treatment of, 593  
   mortality in, 592  
   pain in, 591  
   and rachitis, 589  
   resorptive temperature in, 592  
 Scrofula, 236  
   bones and joints in, 240  
   course and prognosis of, 240  
   diagnosis of, 240  
   ears in, 239  
   etiology of, 236  
   eye in, 239  
   forms of, 230  
   lymph-nodes in, 239  
   morbid anatomy of, 237  
   mucous membranes in, 238  
   occurrence of, 236  
   pyogenic form, morbid anatomy of, 237  
   skin in, 238  
   symptoms of, 238  
   treatment of, 241  
   tuberculous form, morbid anatomy of, 237  
 Scurvy, 588  
 Seborrhoea capillitii, 641  
   treatment of, 645  
 Sepsis, diagnosis of, 97  
   leucocytosis in, 571  
   morbid anatomy of, 97  
   in newborn infant, hemorrhages of, 97



- Sepsis, prognosis of, 98  
treatment of, 98
- Septic infection, 95, 98  
bacteria of, 95  
cryptogenetic, 106  
etiology of, 95  
nervous system in, 96  
skin in, 96  
symptoms of, 96  
umbilicus in, 96
- Septum defects, auricular-ventricular, 441
- Sight, 21, 48  
anomalies of, 22
- Sinus in empyema, 429
- Skin, care of, 639  
desquamation of, in newborn infant, 639  
diffuse induration of, in hereditary syphilis, 267  
infiltration of, in syphilis, 271  
diphtheria of, 218  
dusting-powder for, 45  
eruptions of, in hereditary syphilis, 270  
late syphilitic lesions of, 265
- Skull, deformities of, 527
- Soft palate, paralysis of, 221
- Spasm, congenital gastric, 313
- Spasmus nutans, 498  
etiology of, 499  
symptoms of, 499  
treatment of, 499
- Speech in chorea, 492
- Spina bifida, 528  
course of, 530  
diagnosis of, 531  
lumbalis, 531  
occulta, 531  
spherical and elliptical forms of, 530  
symptoms of, 530  
treatment of, 532  
ventosa, 272
- Spine, anatomical curves, 28  
examination of, 28  
in rachitis, 538
- Spleen in acute leukæmia, 579  
in Buhl's disease, 106  
in catarrhal jaundice, 569  
in cerebrospinal meningitis, 194  
in chronic gastro-enteritis, 569  
in cirrhosis of the liver, 569  
gummata of, 570  
in heart disease, 569  
and kidney, tumors of, 570  
palpation of, 569  
percussion of, 269  
in pseudoleukæmia, 569  
in pseudoleukæmic anæmia, 576  
in rachitis, 569  
in sepsis, 569  
in syphilis, 569
- Spleen in syphilis, congenital, 268  
late, 265  
in typhoid fever, 569
- Sprue, nature of, 284  
occurrence of, 285  
symptoms of, 285  
treatment of, 285
- Sputum, 246
- Starvation, effect of, on breast milk, 60
- Status lymphaticus, 581
- Status prævens, taking of, 20
- Stenosis of pylorus. *See* Hypertrophic pyloric Stenosis.
- Sterilization of milk, disadvantages, 71
- Sterilized milk in scorbutus, 589
- Sterilizer, Arnold's, 71
- Sterility a result of vulvo-vaginitis, 636
- Stethoscope, use of, 22
- Stomacacæ. *See* Ulcerative stomatitis.
- Stomach bacteria, 308  
capacity of, 306  
diphtheria of, 210  
functions and mobility of, 306  
juices, reaction of, 306  
percussion of, 307  
physiological facts, 305  
situation of, 305  
washing of, 39  
indications for, 39  
methods of, 40, 41
- Stomatitis, aphthous, 283  
bacteriology of, 282  
course of, 283  
etiology of, 282  
symptoms of, 282, 283  
gonorrhæal, 289  
symptoms of, 289  
treatment of, 289  
in measles, 153  
pseudodiphtheritic, 288  
treatment of, 288  
in scarlet fever, 129  
toxic, 285  
treatment of, 287  
ulcerative, 287  
etiology of, 287
- Stools of bottle and breast-fed infants, 74  
infant characters of, 308
- Strabismus in cerebrospinal meningitis, 192
- Stridor, congenital, 483
- St. Vitus' dance, 489
- Subphrenic abscess, 431  
diagnosis of, 431  
liver dulness in, 608  
treatment of, 431
- Sucking pads, in atrophy, 327
- Sudamina, 45, 647
- Sydenham's chorea, 489
- Syphilis acquired, 261  
diagnosis of, 262  
etiology of, 261  
prognosis of, 262

- Syphilis acquired, symptoms of, 262  
 anemia in, 572  
 blood in, 271  
 of bones, 264, 271, 595  
 congenital, 265  
   blood in, 271  
   bones in, 271  
   cerebral symptoms in, 272  
   diagnosis of, 273  
   diffuse induration of skin in, 271  
   eye in, 273  
   liver in, 272  
   morbid anatomy of, 266, 267, 268  
   pancreas in, 268  
   prognosis of, 274  
   symptoms of, 268  
   treatment of, 274, 276  
 of the ear, 265  
 hereditary, bone-changes in, 268  
   glandular apparatus in, 268  
   late, 263  
   bone lesions of, 264  
   eye lesions of, 263  
   liver in, 609  
   thymus gland in, 568  
   in wet nurse, 50  
 Syringe, nasal, 37  
 Syringomyelocoele, 529
- T**ACHE cerebrale in cerebrospinal meningitis, 192  
 Tachycardia in diphtheria, 217  
 Tania, 354  
   Bothriocephalus latus, 355  
   elliptica, 354  
   mediocanellata, 355  
   solum, 354  
 Tapeworm, 354  
 Tay-Kingdon spot, 32  
 Teeth, erosions of, 279  
 Temperature, children, 47  
   infant's, mode of taking, 46  
   of the newborn infant, 46  
 Tenesmus in acute, intestinal obstruction, 341  
 Terrors, night, 488  
 Tetanilla, 479  
 Tetanus neonatorum, duration of, 109  
   prognosis of, 109  
   treatment of, 109  
   of the newborn, 108  
 Tetany, 479  
   diagnosis of, 482  
   duration of, 481  
   etiology of, 479  
   forms and frequency of, 479  
   morbid anatomy of, 479  
   mortality of, 482  
   muscular contractures of, 480  
   prognosis of, 482  
   symptoms of, 480  
 Tetany, treatment of, 482  
 Threadworms, 354
- Thrush. *See* Sprue.  
 Thymus gland, abnormal state of, 567  
   carcinoma of, 568  
   diphtheria of, 568  
   hemorrhages in, 568  
   hypertrophy of, 567  
   percussion of, 567  
   sudden death with enlarged thymus, 581  
   syphilis of, 568  
   weight of, 567  
 Thyroid gland, diseases of, 559  
 Tic, forms of, 498  
 Titubation, cerebellar, 32  
 Tongue, desquamation of, 293  
   diseases of, 291  
   ringworm of, 293  
   swallowing, 293  
   treatment of, 294  
 tie, 294  
   treatment of, 294  
 wandering rash of, 291  
   etiology of, 292  
   symptoms of, 292  
   treatment of, 293
- Tonsillitis, otitis in, 598  
 Tonsils, herpes of, 302  
 Torticollis, 20  
 Toxins, action in scorbutus, 589  
 Treatment of adenitis, acute, 558  
   of amygdalitis, acute follicular, 302  
   of ani, prolapsus, 349  
   of aphthae, Bednar's, 284  
   of asphyxia in the newborn infant, 91  
   of atelectasis, 94  
   of bronchiectasis, 373  
   of bronchopneumonia, 404, 407  
     in diphtheria, 234  
   of persistent, 408  
   of bronchitis, acute, 364  
     fibrinous, 366  
   of cholera infantum, 321  
   of chorea insaniens, 498  
   of colic, 313  
   of cretinism, 564  
   of cystitis, 637  
   of dermatitis exfoliativa, 647  
   of diabetes insipidus, 555  
   of dilatation of heart, 466  
   of diphtheroid, 235  
   of dysentery, 329  
   of dyspepsia, acute gastric, 309  
   of eczema, 642  
     squamous, 643  
   of emphysema of lungs, 370  
   of empyema, 426  
     perforating, 425  
   of endocarditis, acute, 449  
   of epilepsy, 488  
   of Erb's palsy, 518  
   of erythema multiform, 645  
   of facial palsy, 515



- Treatment of fatty degeneration, acute,  
in the newborn, 106  
of fissure of anus, 350  
of gastro-enteritis, 321  
in diphtheria, 234  
of gastro-intestinal atrophy, 327  
of glandular fever, 190  
of gonorrhœal stomatitis, 289  
of hæmophilia, 583  
of heart disease, chronic, 454  
of hereditary ataxia, 520  
of hydrocephalus, chronic internal,  
503  
of hysteria, 479  
of ichthyosis, 648  
of icterus neonatorum, 111  
of idiopathic hemorrhage from umbilicus, 103  
of infantile cerebral palsy, 512  
convulsions, 475  
scorbutus, 593  
of inflammation of the mastoid process, 605  
of intertrigo, 644  
of intestinal obstruction, 343  
of jaundice, 611  
of laryngismus stridulus, 486  
of laryngitis, acute catarrhal, 304  
of lobar pneumonia, 389  
of lymphadenitis, chronic, 559  
of melæna neonatorum, 104  
of malarial fever, 183  
of multiple neuritis, 518  
of mumps, 200  
of myocarditis, 458  
of nephritis, acute parenchymatous, 623  
of noma, 291  
of ophthalmia neonatorum, 110  
of osteomyelitis, 598  
of paralysis, acute atrophic, 524  
in diphtheria, 234  
pseudohypertrophic muscular, 527  
of paranephritis, 632  
of pavor nocturnus, 489  
of pemphigus neonatorum, 649  
of pericarditis, 464  
of pertussis, 204, 205  
of pleurisy, dry, 410  
of proctitis, 351  
of pseudodiphtheritic stomatitis, 288  
of pseudoleukæmic anemia, 576  
of pulmonary tuberculosis, 247  
of purpura hæmorrhagica, 585  
rheumatica, 585  
simple, 583  
of rachitis, 543  
of rectal polypi, 352  
of renal calculi, 619  
of retropharyngeal abscess, acute, 297  
of rheumatism, acute articular, 550  
of rheumatoid arthritis, 545
- Treatment of rōtheln, 143  
of scrofula, 241  
of sepsis, 98  
of spina bifida, 532  
of sprue, 285  
of subphrenic abscess, 431  
of syphilis, congenital, 274, 276  
of tetanus neonatorum, 109  
of tetany, 482  
of tongue-swallowing, 29  
of tongue-tie, 294  
of toxic stomatitis, 287  
of tuberculosis of peritoneum, 251  
of tuberculous meningitis, 261  
of tympanites, 513  
of umbilical fungus, 99  
hernia, 103  
phlebitis, 101  
of varicella, 162  
of vulvovaginitis, 636  
of wandering rash of tongue, 293
- Trismus neonatorum, 108  
Trousseau's phenomenon, 581  
in tuberculous meningitis, 255
- Tubercle, solitary, of brain, 261  
Tuberculous meningitis, Babinski's reflex  
in, 257
- Tuberculosis, abdominal, 252  
anæmia in, 572  
avenues of infection, 242  
cows' milk, 242  
intestinal canal, 242  
mother's milk, 242  
placental, 242  
ritual circumcision, 242  
through wounds, 242  
of bones, 595  
of brain, 261  
and diphtheria, 219  
etiology of, 241  
frequency of, 243  
of kidney, 630  
of larynx, 251  
morbid anatomy of, 244  
of peritoneum, 247  
adhesions in, 250  
constipation in, 248  
course of, 251  
diagnosis of, 250  
etiology of, 247  
liver in, 249  
miliary nodular adhesive form,  
247  
morbid anatomy of, 247  
occurrence of, 247  
pain in, 248  
physical signs of, 249  
spleen in, 249  
symptoms of, 248  
temperature in, 248  
treatment of, 251  
vomiting in, 249  
pulmonary, 243

- Tuberculosis, pulmonary, course of, 246  
 diagnosis of, 246  
 hæmoptysis in, 246  
 localization of, 244  
 symptoms of, 245  
 temperature in, 246  
 treatment of, 247  
 in scarlet fever, 133  
 statistics, 242  
 in the wet nurse, 50
- Tuberculous meningitis. *See* Meningitis.  
 choroid, tubercle of, 259  
 cries, hydrocephalic in, 257  
 facial palsies in, 256  
 hippus in, 255  
 vomiting in, 254  
 osteomyelitis, 598
- Tubes for intubation, retained, 233
- Tumor in acute intestinal obstruction, 341  
 contour of abdomen in tumor, 26  
 of kidney and spleen, 590  
 polypoid, of the gut, 27
- Tympanites, 313  
 in bronchopneumonia, 390  
 treatment, 313
- Tympanitic distention, in intestinal perforation, 26  
 in peritonitis, 26  
 in typhoid fever, 26
- Tympanum spontaneous rupture of, 600
- Typhoid fever, 166  
 blood in, 173  
 bronchopneumonia in, 398  
 cerebral invasion in, 168  
 complications of, 174  
 cystitis in, 637  
 Diazo reaction in, 176  
 diet in, 178  
 and diphtheria, 219  
 enlarged spleen in, 170  
 fetal, 167  
 frequency, 166  
 heart in, 173  
 hæmorrhages in, 172  
 hydrotherapy in, 178  
 infantile, 167, 169, 170  
 lungs in, 173  
 morbid anatomy of, 167  
 nervous symptoms of, 173  
 occurrence of, 166  
 otitis in, 173, 598  
 parotitis in, 173  
 petechiæ, 170  
 pneumonic invasion in, 168  
 prognosis of, 177  
 relapses in, 174  
 roseola of, 170  
 sequelæ, skin, 174  
 symptoms of, 168  
 treatment of, 178  
 alcohol in, 178  
 heart in, 178  
 hæmorrhages, 178
- Typhoid fever, ulcerations of the gut in, 167  
 variety of cases, 168  
 Widal reaction in, 176
- Typhoidal ulcer, hæmorrhage from, 343
- U**MBILICUS, diseases of, 98  
 fungus of, 99  
 hæmorrhage from, 101  
 infection in the newborn infant, morbidly, 17  
 in septic infection, 96
- Uræmia in nephritis, 622, 623  
 in scarlet fever, 131
- Urethritis, 636  
 gonorrhæal, 636  
 simple, 636
- Urine, 53, 614  
 in carcinoma of kidney, 629  
 in chorea, 492  
 discolorations of, 19  
 indican in, 615  
 in lithiasis, 19  
 in pertussis, 203  
 quantity of, 615  
 reaction in cystitis, 637  
 in sarcoma of kidney, 628
- Urogenital blennorrhœa, 633
- V**ACCINATION, 162  
 age of, 163  
 complications of, 165  
 course of, 164  
 eruptions in, 166  
 bullous, 166  
 erythematous, 166  
 morbilliform, 166  
 pemphigoid, 166  
 pustular, 166  
 scarlatinoid, 166  
 urticarial, 166  
 etiology of, 167  
 fever in, 164  
 and hæmorrhagic diathesis, 163  
 human virus, 163  
 inoculation of the eye, 166  
 lymph of, 164  
 mode of, 163  
 osteomyelitis in, 165  
 sepsis in, 165  
 tetanus in, 165
- Vaccinia, generalized, 165
- Varicella, bronchopneumonia in, 398  
 bullosa, 162  
 and cardiac hypertrophy and dilatation, 161  
 complications of, 161  
 definition of, 159  
 diagnosis of, 161  
 enanthema of, 160  
 exanthema of, 159  
 and fatty liver, 161  
 gangrene of the skin in, 161



- Varicella, incubation of, 159
  - joint-affections in, 161
  - nephritis in, 161
  - otitis in, 598
  - prognosis of, 162
  - rheumatoid affections in, 161
  - symptoms of, 159
  - temperature in, 160
  - treatment of, 162
  - and variola, 161
  - and varioloid, 161
- Ventricle, left, marking out dulness of, 435
- Ventricular septum, congenital deficiency, 441
- Vesiculo-tympanic resonance in atelectasis, 93
- Vincent's spirillum, 287
- Viscera, displacement of, in pleuritic effusion, 420
- Vomiting, forms of, 19, 311
  - habitual, 310
  - in scarlet fever, 132
  - in tuberculous meningitis 254
  - ushering in illness, 19
- Vulva, diphtheria of, 218
- Vulvovaginitis, 633
  - arthritis in, 636
  - course and complications of, 636
  - in diphtheria, treatment of, 234
  - etiology of, 634
- Vulvovaginitis, forms of, 633, 634
  - symptoms of, 635
  - treatment of, 636
- W**ALK, spastic, 32
- Weaning breast-fed infants, 81
- Weight, average, of infants and children, 55
  - curve of loss of weight in the new-born infant, 54
  - daily increase of, 53, 55, 66
  - loss in first three days, 53
  - normal daily increase, curve of, 55
- Weil's disease, 610
- Wet nurse, mental state of, 50
  - milk examination of, 50
  - quality of milk, 50
  - quantity of milk, 50
  - selection of, 49
    - age of, 49
    - breasts of, 49
    - examination of, 49
    - nipple, of breasts, 49
- Whey, method of milk dilution, 68
  - proteids, 59
- White blood cells, 571
- Whooping-cough. *See* Pertussis.
- Woodward's buret for proteids, 63
- Worms, round, 353
  - tape, 354
  - thread, 354

U. S. A.

Arthur L. Gilbert.



# Catalogue of Books

PUBLISHED BY

## Lea Brothers & Company,

706, 708 & 710 Sansom St., Philadelphia.  
111 Fifth Avenue, New York.



Our publications can be purchased from any Bookseller in the United States or Canada, or they will be delivered by express or mail, carriage paid to any address on receipt of the printed price.

JULY, 1903.

### STANDARD MEDICAL PERIODICALS.

#### Progressive Medicine.

A Quarterly Digest of New Methods, Discoveries and Improvements in the Medical and Surgical Sciences by Eminent Authorities. Edited by DR. HOBART AMORY HARE. In four abundantly illustrated, cloth bound, octavo volumes of 400-500 pages each, issued quarterly, commencing with March each year. Per annum (4 volumes), \$10.00, delivered.

#### The Medical News.

WEEKLY, \$4.00 PER ANNUM.

Each number contains 48 quarto pages, abundantly illustrated. A crisp, fresh weekly professional newspaper.

#### The American Journal of the Medical Sciences.

MONTHLY, \$5.00 PER ANNUM.

Each issue contains 192 octavo pages, fully illustrated. The most advanced, practical, original and enterprising American exponent of scientific medicine.

#### The Medical News Visiting List for 1903.

Four styles, Weekly (dated for 30 patients); Monthly (undated, for 120 patients per month); Perpetual (undated, for 30 patients weekly per year); and Perpetual (undated, for 60 patients weekly per year). Each style in one wallet-shaped book, leather bound, with pocket, pencil and rubber. Price, each, \$1.25. Thumb-letter index, 25 cents extra.

#### The Medical News Pocket Formulary.

New (5th) edition carefully revised to date. Containing 1700 prescriptions representing the latest and most approved methods of administering remedial agents. Strongly bound in leather, with pocket and pencil. Price, \$1.50, net.

#### COMBINATION RATES.

	ALONE.	IN COMBINATION
Per Annum American Journal of the Medical Sciences.....	\$5.00	\$8.00 } \$16.00
Medical News.....	4.00	
Progressive Medicine.....	10.00	
Medical News Visiting List.....	1.25	
Medical News Formulary.....	1.50, net.	

In all \$21.75 for \$17.00

First four above publications in combination.....\$16.75

All above publications in combination..... 17.00

Other Combinations will be quoted on request. Full Circulars and Specimen

**ABBOTT (A. C.).** *PRINCIPLES OF BACTERIOLOGY*: a Practical Manual for Students and Physicians. Sixth edition enlarged and thoroughly revised. 12mo., 636 pages, with 111 engravings, of which 26 are colored. Cloth, \$2.75, *net*.

**ALLEN (CHARLES W.).** *A TREATISE ON RADIOTHERAPY AND PHOTOTHERAPY*. Octavo, about 500 pages, with many illustrations. *Shortly*.

**ALLEN (HARRISON).** *A SYSTEM OF HUMAN ANATOMY*. Comprising 813 quarto pages, with 380 engravings on stone and 241 woodcuts. Cloth, \$23.

**AMERICAN SYSTEM OF PRACTICAL MEDICINE.** In Contributions by Eminent American Authors. Edited by ALFRED L. LOOMIS, M.D., LL.D., and W. GILMAN THOMPSON, M.D. In four octavo volumes of about 900 pages each, fully illustrated. Per volume, cloth, \$5; leather, \$6; half morocco, \$7. *For sale by subscription only.* Prospectus free on application.

**AMERICAN SYSTEM OF DENTISTRY. IN TREATISES BY VARIOUS AUTHORS.** Edited by WILBUR F. LITCH, M.D., D.D.S. In three octavo volumes, containing about 3200 pages, with 1873 illustrations and many full-page plates. Per volume, cloth, \$6; leather, \$7. Prospectus free on application to the Publishers.

**AMERICAN TEXT-BOOK OF ANATOMY.** See *Gerrish*, page 7.

**AMERICAN TEXT-BOOKS OF DENTISTRY. IN CONTRIBUTIONS BY EMINENT AMERICAN AUTHORITIES.** In two octavo volumes of more than 800 pages each, richly illustrated:

—— *PROSTHETIC DENTISTRY.* Edited by CHARLES J. ESSIG, M.D., D.D.S., Professor of Mechanical Dentistry and Metallurgy, Department of Dentistry, University of Pennsylvania, Philadelphia. Second Edition, 807 pages, with 1089 engravings. Cloth, \$6; leather, \$7, *net*.

—— *OPERATIVE DENTISTRY.* Edited by EDWARD C. KIRK, D.D.S., Professor of Clinical Dentistry, Department of Dentistry, University of Pennsylvania. Second Edition, 857 pages, 897 engravings. Cloth, \$6.00; leather, \$7.00, *net*.

**AMERICAN SYSTEMS OF GYNECOLOGY AND OBSTETRICS.** Gynecology edited by MATTHEW D. MANN, A.M., M.D., and Obstetrics edited by BARTON C. HIRST, M.D. In four large octavo volumes comprising 3612 pages, with 1092 engravings, and 8 colored plates. Per volume, cloth, \$5.

**ARCHINARD (P. E.).** *AN EPITOME OF BACTERIOLOGY.* 12mo., about 240 pages, with many illustrations. Cloth, \$1.00, *net*. *Lea's Series of Medical Epitomes.* See page 10.

**ASHHURST (JOHN, JR.).** *THE PRINCIPLES AND PRACTICE OF SURGERY.* For the use of Students and Practitioners. Sixth and revised edition. In one 8vo. volume of 1161 pages, with 656 engravings. Cloth, \$6; leather, \$7.

**A SYSTEM OF PRACTICAL MEDICINE BY AMERICAN AUTHORS.** Edited by WILLIAM PEPPER, M.D., LL.D. In five large octavo volumes, containing 5573 pages and 198 illustrations. Price per volume, cloth, \$5. Prospectus free on application.

**A TREATISE ON SURGERY BY AMERICAN AUTHORS. FOR STUDENTS AND PRACTITIONERS OF SURGERY AND MEDICINE.** Edited by ROSEWELL PARK, M.D. Third edition. Octavo, 1408 pages, with 692 engravings and 64 plates. Cloth, \$7.00, *net*; leather, \$8.00, *net*. ~~\$9.50~~ Published also in 2 volumes at \$3.75 *net* per vol. for cloth.

**ATTFIELD (JOHN).** *CHEMISTRY; GENERAL, MEDICAL AND PHARMACEUTICAL.* Sixteenth edition, specially revised by the Author for America. In one handsome 12mo. volume of 784 pages, with 88 illustrations. Cloth, \$2.50, *net*.



**BACON (GORHAM).** *ON THE EAR.* Third Edition. One 12mo. volume, 430 pages, with 120 engravings and 7 colored plates. Cloth, \$2.25, *net*.

**BALLENGER (W. L.) AND WIPPERN (A. G.).** *A POCKET TEXT-BOOK OF DISEASES OF THE EYE, EAR, NOSE AND THROAT.* 12mo., 525 pages, with 148 illustrations, and 6 colored plates. Cloth, \$2, *net*. Flexible red leather, \$2.50, *net*.

**BARNES (ROBERT AND FANCOURT).** *A SYSTEM OF OBSTETRIC MEDICINE AND SURGERY.* Octavo, 872 pages, 231 illustrations. Cloth, \$5.

**BARTHOLOW (ROBERTS).** *CHOLERA; ITS CAUSATION, PREVENTION AND TREATMENT.* 12mo., 127 pages, with 9 illustrations. Cloth, \$1.25.

**BILLINGS (JOHN S.).** *THE NATIONAL MEDICAL DICTIONARY.* Including in one alphabet English, French, German, Italian and Latin Technical Terms used in Medicine and the Collateral Sciences. In two imperial octavo volumes, containing 1574 pages and two colored plates. Per vol., leather, \$7. Specimen pages on application.

**BLACK (D. CAMPBELL).** *THE URINE IN HEALTH AND DISEASE, AND URINARY ANALYSIS, PHYSIOLOGICALLY AND PATHOLOGICALLY CONSIDERED.* 12mo., 256 pages, 73 engravings. Cloth, \$2.75.

**BLOODGOOD (JOSEPH C.).** *A TREATISE ON THE PRINCIPLES OF SURGERY.* Octavo about 1000 pages, with many engravings and plates in black and colors. *Preparing.*

**BLOXAM (C. L.).** *CHEMISTRY, INORGANIC AND ORGANIC.* With Experiments. New American from the fifth London edition. In one handsome octavo volume of 727 pages, with 292 illustrations. Cloth, \$2; leather, \$3.

**BREWER (GEORGE E.).** *A TEXT-BOOK OF THE PRINCIPLES AND PRACTICE OF SURGERY.* Octavo, 700 pages, with 280 engravings and 7 plates. Cloth, \$4.00; leather, \$5.00, *net*. *Just Ready.*

**BRUCE (J. MITCHELL).** *MATERIA MEDICA AND THERAPEUTICS.* Sixth edition. In one 12mo. volume of 600 pages. Cloth, \$1.50, *net*. *Students' Series of Manuals.* Page 15.

— *PRINCIPLES OF TREATMENT.* In one octavo volume of 625 pages. Cloth, \$3.75, *net*.

**BRYANT (THOMAS).** *THE PRACTICE OF SURGERY.* Fourth edition. Octavo, 1040 pages, with 727 illustrations. Cloth, \$6.50; leather, \$7.50.

**BURCHARD (HENRY H.).** *DENTAL PATHOLOGY AND THERAPEUTICS, INCLUDING PHARMACOLOGY.* Handsome octavo, 575 pages, with 400 illustrations. Cloth, \$5; leather, \$6, *net*.

**BURNETT (CHARLES H.).** *A TREATISE ON THE EAR.* Second edition. 8vo., 580 pages, with 107 illustrations. Cloth, \$4; leather, \$5.

**CARTER (R. BRUDENELL) AND FROST (W. ADAMS).** *OPHTHALMIC SURGERY.* In one pocket-size 12mo. volume of 559 pages, with 91 engravings and one plate. Cloth, \$2.25. See *Series of Clinical Manuals*, page 13.

**CASPARI (CHARLES, JR.).** *A TREATISE ON PHARMACY.* For Students and Pharmacists. Second edition. Revised and enlarged. Octavo, 774 pages, with 302 illustrations. Cloth, \$4.25, *net*.

**CHAPMAN (HENRY C.).** *HUMAN PHYSIOLOGY.* Second edition. Octavo, 921 pages, with 595 illustrations. Cloth, \$4.25; leather, \$5.25, *net*.

**CHARLES (T. CRANSTOUN).** *PHYSIOLOGICAL AND PATHOLOGICAL CHEMISTRY.* Octavo, 451 pages, with 38 engravings and 1 colored plate. Cloth, \$3.50.

**CHEYNE (W. WATSON) AND BURGHARD (F. F.).** *SURGICAL TREATMENT.* In seven octavo cloth bound volumes, illustrated. Volume I, 299 pages and 66 engravings; \$3.00, *net*. Volume II, 382 pages, 141 engravings; \$4.00, *net*. Volume III, 300 pages, 100 engravings; \$3.50, *net*. Volume IV, 383 pages, 138 engravings; \$3.75, *net*. Volume V, 482 pages, 145 engravings; \$5.00, *net*. Vol. VI, 498 pages, 124 engravings; \$5.00, *net*. Volume VII, \$5.75, *net*. *Complete work Just Ready.*

**CLELAND (JOHN).** *A DIRECTORY FOR THE DISSECTION OF THE HUMAN BODY.* In one 12mo. volume of 178 pages. Cloth, \$1.25.

**CLOUSTON (THOMAS S.).** *CLINICAL LECTURES ON MENTAL DISEASES.* New (5th) edition. Crown 8vo., of 736 pages with 19 colored plates. Cloth, \$4.25, *net*. FOLSOM's *Abstract of Laws of U.S. on Custody of Insane*, octavo, \$1.50, is sold in conjunction with *Clouston on Mental Diseases* for \$5.00, *net*, for the two works.

**COAKLEY (CORNELIUS G.).** *DISEASES OF THE NOSE, THROAT, NASOPHARYNX AND TRACHEA.* Second edition. 12mo., 556 pages, with 103 engravings, and 4 colored plates. Cloth, \$2.75, *net*.

**COATS (JOSEPH).** *A TREATISE ON PATHOLOGY.* Octavo, 829 pages, with 339 engravings. Cloth, \$5.50; leather, \$6.50.

**COLEMAN (ALFRED).** *A MANUAL OF DENTAL SURGERY AND PATHOLOGY.* Octavo, 412 pages, with 331 engravings. Cloth, \$3.25.

**COLLINS (H. D.) AND ROCKWELL (W. H., JR.).** *A POCKET TEXT-BOOK OF PHYSIOLOGY.* 12mo., of 316 pages, with 153 illustrations. Cloth, \$1.50, *net*; flexible leather, \$2.00, *net*. *Lea's Series of Pocket Text Books.* Page 12.

**CONDIE (D. FRANCIS).** *A PRACTICAL TREATISE ON THE DISEASES OF CHILDREN.* Sixth edition. 8vo. 719 pages. Cloth, \$5.25; leather, \$6.25.

**CORNIL (V.).** *SYPHILIS: ITS MORBID ANATOMY, DIAGNOSIS AND TREATMENT.* Translated, by J. HENRY C. SIMES, M.D., and J. WILLIAM WHITE, M.D. 8vo. 461 pages, with 84 illustrations. Cloth, \$3.75.

**CROCKETT (M. A.).** *A POCKET TEXT-BOOK OF DISEASES OF WOMEN.* 12mo. of 368 pages, with 107 illustrations. Cloth, \$1.50, *net*. Flexible Red Leather, \$2.00, *net*. *Lea's Series of Pocket Text-Books.* Page 12.

**CROOK (JAMES K.).** *MINERAL WATERS OF UNITED STATES.* Octavo, 574 pages. Cloth, \$3.50, *net*.

**CULBRETH (DAVID M. R.).** *MATERIA MEDICA AND PHARMACOLOGY.* Third edition. Octavo, 905 pages, with 473 engravings. Cloth, \$4.75, *net*.

**CUSHNY (ARTHUR R.).** *PHARMACOLOGY AND THERAPEUTICS.* Third edition. Octavo of 750 pages, with 52 illustrations. Cloth, \$3.75; leather, \$4.75, *net*.

**DALTON (JOHN C.).** *A TREATISE ON HUMAN PHYSIOLOGY.* Seventh edition, thoroughly revised. Octavo of 722 pages, with 252 engravings. Cloth, \$5; leather, \$6.



- DALTON (JOHN C.). DOCTRINES OF THE CIRCULATION OF THE BLOOD.** In one handsome 12mo. volume of 293 pages. Cloth, \$2.
- DAVENPORT (F. H.). DISEASES OF WOMEN.** Fourth edition. 12mo., 402 pages and 154 engravings. Cloth, \$1.75, *net*.
- DAVIS (F. H.). LECTURES ON CLINICAL MEDICINE.** Second edition. In one 12mo. volume of 287 pages. Cloth, \$1.75.
- DAVIS (EDWARD P.). A TREATISE ON OBSTETRICS.** Octavo, 546 pages, with 217 engravings, and 30 full-page plates in colors and monochrome. Cloth, \$5; leather, \$6.
- DE LA BECHE'S GEOLOGICAL OBSERVER.** In one large octavo volume of 700 pages, with 300 engravings. Cloth, \$4.
- DENNIS (FREDERIC S.) AND BILLINGS (JOHN S.). A SYSTEM OF SURGERY.** In Contributions by American Authors. In four very handsome octavo volumes, containing 3652 pages, with 1585 engravings, and 45 full-page plates in colors and monochrome. Per volume, cloth, \$6; leather, \$7; half morocco, gilt back and top, \$8.50. *For sale by subscription only.* Full prospectus free.
- DERCUM (FRANCIS X.). Editor. A TEXT-BOOK ON NERVOUS DISEASES.** By American Authors. Octavo, 1054 pages, with 341 engravings and 7 colored plates.
- DE SCHWEINITZ (GEORGE E.). THE TOXIC AMBLYOPIAS.** Very handsome octavo, 240 pages, 46 engravings, and 9 full-page plates in colors. De luxe binding, \$4, *net*.
- DRAPER (JOHN C.). MEDICAL PHYSICS.** A Text-book for Students and Practitioners of Medicine. Octavo of 734 pages, with 376 engravings. Cloth, \$4.
- DRUITT (ROBERT). THE PRINCIPLES AND PRACTICE OF MODERN SURGERY.** From the twelfth London edition, edited by STANLEY BOYD, F.R.C.S. Large octavo, 965 pages, with 373 engravings. Cloth, \$4; leather, \$5.
- DUANE (ALEXANDER). A DICTIONARY OF MEDICINE AND THE ALLIED SCIENCES.** Comprising the Pronunciation, Derivation and Full Explanation of Medical, Dental, Pharmaceutical and Veterinary Terms. Together with much Collateral Descriptive Matter, Numerous Tables, etc. Fourth edition, with Appendix. Square octavo volume of 688 pages with 8 colored plates and thumb index. Cloth, \$3.00, *net*; limp leather, \$4.00, *net*.
- DUDLEY (E. C.). PRINCIPLES AND PRACTICE OF GYNECOLOGY.** Third edition. Octavo, 756 pages, with 474 engravings, of which 60 are colored, and 22 full page plates in colors and monochrome. Cloth, \$5.00, *net*; leather, \$6.00, *net*; half morocco, \$6.50, *net*.
- DUNGLISON (ROBLEY). A DICTIONARY OF MEDICAL SCIENCE.** Containing a full Explanation of the Various Subjects and Terms of Anatomy, Physiology, Medical Chemistry, Pharmacy, Pharmacology, Therapeutics, Medicine, Hygiene, Dietetics, Pathology, Surgery, Ophthalmology, Otology, Laryngology, Dermatology, Gynecology, Obstetrics, Pediatrics, Medical Jurisprudence, Dentistry, Veterinary Science, etc., etc. By ROBLEY DUNGLISON, M.D., LL.D., late Professor of Institutes of Medicine in the Jefferson Medical College of Philadelphia. Edited by RICHARD J. DUNGLISON, A.M., M.D. Twenty-second edition, thoroughly revised and greatly enlarged and improved, with the Pronunciation, Accentuation and Derivation of the Terms. With Appendix. Imperial octavo of 1350 pages, with thumb letter index. Cloth, \$7.00, *net*; leather, \$8.00, *net*. *This edition contains portrait of Dr. Robley Dunglison.*
- DUNHAM (EDWARD K.). MORBID AND NORMAL HISTOLOGY.** Octavo, 450 pages, with 360 illustrations. Cloth, \$3.25, *net*.

- DUNHAM (EDWARD K.).** *NORMAL HISTOLOGY.* Second edition. Octavo, 319 pages, with 244 illustrations. Cloth, \$2.50, *net*.
- DWIGHT (E. W.).** *AN EPITOME OF MEDICAL JURISPRUDENCE.* 12mo., about 240 pages. Cloth, \$1.00, *net.* *Lea's Series of Medical Epitomes.* See page 10.
- *AN EPITOME OF TOXICOLOGY.* 12mo., about 225 pages. Cloth, \$1.00, *net.* *Lea's Series of Medical Epitomes.* See page 10.
- ECKLEY (WILLIAM T.).** *A GUIDE TO DISSECTION OF THE HUMAN BODY.* Octavo, 400 pages, with 220 large engravings in black and colors. Cloth, \$3.50, *net.*
- *REGIONAL ANATOMY OF THE HEAD AND NECK FOR STUDENTS AND PRACTITIONERS.* Octavo, 240 pages, with 36 engravings and 20 plates in black and colors. Cloth, \$2.50, *net.*
- EDES (ROBERT T.).** *TEXT-BOOK OF THERAPEUTICS AND MATERIA MEDICA.* In one 8vo. volume of 544 pages. Cloth, \$3.50; leather, \$4.50.
- EDIS (ARTHUR W.).** *DISEASES OF WOMEN.* 8vo., 576 pages, with 148 engravings. Cloth, \$3; leather, \$4.
- EGBERT (SENECA).** *HYGIENE AND SANITATION.* Second edition. In one 12mo. volume of 427 pages, with 77 illustrations. Cloth, \$2.25, *net.*
- ELLIS (GEORGE VINER).** *DEMONSTRATIONS IN ANATOMY.* Eighth edition. Octavo, 716 pages, with 249 engravings. Cloth, \$4.25; leather, \$5.25.
- EMMET (THOMAS ADDIS).** *THE PRINCIPLES AND PRACTICE OF GYNÆCOLOGY.* For the use of Students and Practitioners. Third edition, enlarged and revised. 8vo. of 880 pages, with 150 original engravings. Cloth, \$5; leather, \$6.
- ERICHSEN (JOHN E.).** *THE SCIENCE AND ART OF SURGERY.* From the eighth enlarged and revised London edition. In two large octavo volumes containing 2316 pages, with 984 engravings. Cloth, \$9; leather, \$11.
- ESSIG (CHARLES J.).** *PROSTHETIC DENTISTRY.* Second Edition. See *American Text-books of Dentistry*, page 2.
- ESSIG (C. J.) AND KOENIG (AUGUSTUS).** *DENTAL METALLURGY.* Fourth edition. 12mo. 277 pages with 143 engravings. Cloth, \$1.75, *net.*
- EVANS (DAVID J.).** *A POCKET TEXT-BOOK OF OBSTETRICS.* 12mo. of 409 pages, with 148 illustrations. Cloth, \$1.75, *net*; limp leather, \$2.25, *net.* *Lea's Series of Pocket Text-Books.* Page 12.
- EWING (JAMES).** *A PRACTICAL TREATISE ON THE BLOOD.* Octavo, 432 pages, with 30 engravings and 14 full-page colored plates. Cloth, \$3.50, *net.*
- FARQUHARSON (ROBERT).** *A GUIDE TO THERAPEUTICS.* Fourth edition, revised by FRANK WOODBURY, M.D. 12mo., 581 pages. Cloth, \$2.50.
- FIELD (GEORGE P.).** *A MANUAL OF DISEASES OF THE EAR.* Fourth edition. Octavo, 391 pages, with 73 engravings and 21 colored plates. Cloth, \$3.75.
- FINDLEY (PALMER D.).** *A TREATISE ON GYNÆCOLOGICAL DIAGNOSIS.* Octavo, 493 pages, 210 engravings, 45 plates in black and colors. Cloth, \$4.50; leather, \$5.50, *net.* *Just Ready.*
- FLINT (AUSTIN).** *A TREATISE ON THE PRINCIPLES AND PRACTICE OF MEDICINE.* Seventh edition, thoroughly revised by FREDERICK P. HENRY, M.D. In one large 8vo. volume of 1143 pages, with engravings. Cloth, \$5; leather, \$6.



- FLINT (AUSTIN).** *A PRACTICAL TREATISE ON THE HEART.* Second edition, enlarged. Octavo, 550 pages. Cloth, \$4.
- *MEDICAL ESSAYS.* In one 12mo. volume of 210 pages. Cloth, \$1.38.
- *ON THE CHEST AND RESPIRATORY ORGANS.* Second and revised edition. Octavo, 591 pages. Cloth, \$4.50.
- *ON PHTHISIS.* In one 8vo. volume of 442 pages. Cloth, \$3.50.
- FOLSOM (C. F.).** *AN ABSTRACT OF STATUTES OF U. S. ON CUSTODY OF THE INSANE.* In one 8vo. volume of 108 pages. Cloth, \$1.50.
- FORMULARY, THE NATIONAL.** See *National Dispensatory.* Page 14.
- FORMULARY, POCKET.** Fifth edition. See page 1. \$1.50, net.
- FOSTER (MICHAEL).** *A TEXT-BOOK OF PHYSIOLOGY.* Sixth and revised edition. Octavo, 923 pages, with 257 illustrations. Cloth, \$4.50; leather, \$5.50.
- FOTHERGILL (J. MILNER).** *HAND-BOOK OF TREATMENT.* Third edition. Octavo, 664 pages. Cloth, \$3.75; leather, \$4.75.
- FOWNES (GEORGE).** *A MANUAL OF ELEMENTARY CHEMISTRY.* Twelfth edition. Embodying WATTS' *Physical and Inorganic Chemistry.* Royal 12mo., 1061 pages, with 168 engravings, and 1 colored plate. Cloth, \$2.75; leather, \$3.25.
- FRANKLAND (E.) AND JAPP (F. R.).** *INORGANIC CHEMISTRY.* Octavo, 677 pages, with 51 engravings and 2 plates. Cloth, \$3.75; leather, \$4.75.
- FULLER (EUGENE).** *DISORDERS OF THE SEXUAL ORGANS IN THE MALE.* Octavo, 238 pages, with 25 engravings and 8 plates. Cloth, \$2.
- GALLAUDET (BERN B.).** *A POCKET TEXT-BOOK OF SURGERY.* 12mo. of about 500 pages, with many illustrations. *In Press.* *Lea's Series of Pocket Text-Books.* Page 12.
- GANT (FREDERICK JAMES).** *THE STUDENT'S SURGERY.* A Multum in Parvo. In one square octavo volume of 845 pages, with 159 engravings. Cloth, \$3.75.
- GAYLORD (HARVEY R.) AND ASCHOFF (LUDWIG).** *PATHOLOGICAL HISTOLOGY.* With introductory note by WILLIAM H. WELCH, M.D. Quarto, 354 pages, with 81 engravings and 40 full-page plates. Cloth, \$7.50, net.
- GERRISH (FREDERIC H.).** *A TEXT-BOOK OF ANATOMY.* By American Authors. Edited by FREDERIC H. GERRISH, M.D. Second edition. Imp. octavo, 937 pages, with 1003 illustrations in black and colors. Cloth, \$6.50; sheep, \$7.50, net; half morocco, \$8.00, net.
- GIBBES (HENEAGE).** *PRACTICAL PATHOLOGY AND MORBID HISTOLOGY.* Octavo of 314 pages, with 60 illustrations, mostly photographic. Cloth, \$2.75.
- GRAY (HENRY).** *ANATOMY, DESCRIPTIVE AND SURGICAL.* Fifteenth edition, thoroughly revised. In one imperial octavo volume of 1249 pages, with 780 large and elaborate engravings. Price with illustrations in colors, cloth, \$6.25, net; leather, \$7.25, net. Price, with illustrations in black, cloth, \$5.50; leather, \$6.50, net.
- GRAYSON (CHARLES P.).** *DISEASES OF THE THROAT, NOSE, AND ASSOCIATED AFFECTIONS OF THE EAR.* Octavo, 548 pages, with 129 engravings and 8 plates in colors and monochrome. Cloth, \$3.50, net.
- GREEN (T. HENRY).** *PATHOLOGY AND MORBID ANATOMY.* Ninth revised edition. Octavo, 565 pages, with 339 engravings and 4 colored plates. Cloth, \$3.25, net.
- GREENE (WILLIAM H.).** *MEDICAL CHEMISTRY.* 12mo., 310 pages, with 74 illustrations. Cloth, \$1.75.

**GRINDON (JOSEPH).** *A POCKET TEXT-BOOK OF SKIN DISEASES.* 12mo. of 367 pages, with 39 illustrations. Cloth, \$2.00; flexible leather, \$2.50, *net*. *Lea's Series of Pocket Text-Books.* Page 12.

**GROSS (SAMUEL D.).** *THE URINARY BLADDER, THE PROSTATE GLAND AND THE URETHRA.* Third edition, revised by SAMUEL W. GROSS, M.D. Octavo of 574 pages, with 170 illustrations. Cloth, \$4.50.

**GUENTHER (A. E. AND T. C.).** *AN EPITOME OF PHYSIOLOGY.* 12mo., 225 pages, illustrated. Cloth, \$1.00, *net*. *Lea's Series of Medical Epitomes.* See page 10.

**HABERSHON (S. O.).** *DISEASES OF THE ABDOMEN.* Second American from the third English edition. Octavo, 554 pages, with 11 engravings. Cloth, \$3.50.

**HALL (WINFIELD S.).** *TEXT-BOOK OF PHYSIOLOGY.* Octavo, 672 pages, with 343 engravings and 6 colored plates. Cloth, \$4.00, *net*; leather, \$5.00, *net*.

**HAMILTON (ALLAN McLANE).** *NERVOUS DISEASES.* Second and revised edition. Octavo, 598 pages, with 72 engravings. Cloth, \$4.

**HARDAWAY (W. A.).** *MANUAL OF SKIN DISEASES.* Second edition. 12mo., 560 pages with 40 illustrations and 2 colored plates. Cloth, \$2.25, *net*.

**HARE (HOBART AMORY).** *A TEXT-BOOK OF PRACTICAL THERAPEUTICS,* with Special Reference to the Application of Remedial Measures to Disease and their Employment upon a Rational Basis. Ninth revised edition. Octavo, 851 pages, 105 engravings, 4 colored plates. Cloth, \$4.00, *net*; leather, \$5.00, *net*; half morocco, \$5.50, *net*.

— *PRACTICAL DIAGNOSIS.* The Use of Symptoms in the Diagnosis of Disease. Fifth edition, revised and enlarged. Octavo, 727 pages, with 236 engravings, and 25 full-page plates. Cloth, \$5.00; leather, \$6.00; half morocco, \$6.50, *net*.

— *Editor. A SYSTEM OF PRACTICAL THERAPEUTICS.* By American and Foreign Authors. Second edition. In three large octavo volumes containing 2593 pages, with 457 engravings and 26 full-page plates. Price per volume, cloth, \$5.00, *net*; leather, \$6.00, *net*; half morocco, \$7.00, *net*. *For sale by subscription only.* Full prospectus free on application to the publishers.

— *ON THE MEDICAL COMPLICATIONS AND SEQUELÆ OF TYPHOID FEVER.* Octavo, 276 pages, 21 engravings, and 2 full-page plates. Cloth, \$2.40, *net*.

**HARRINGTON (CHARLES).** *A TREATISE ON PRACTICAL HYGIENE.* Second edition. Handsome octavo of 755 pages, with 113 engravings and 12 plates in colors and monochrome. Cloth, \$4.25, *net*.

**HARTSHORNE (HENRY).** *ANATOMY AND PHYSIOLOGY.* 12mo., 310 pages, with 220 engravings. Cloth, \$1.75.

— *A CONSPECTUS OF THE MEDICAL SCIENCES.* Comprising Manuals of Anatomy, Physiology, Chemistry, Materia Medica, Practice of Medicine, Surgery and Obstetrics. Second edition. 12mo., 1028 pages, with 477 illustrations. Cloth, \$4.25; leather, \$5.

**HAYDEN (JAMES R.).** *A POCKET TEXT-BOOK OF VENEREAL DISEASES.* Third edition. In one 12mo. volume of 304 pages, with 66 engravings. Cloth, \$1.75, *net*; Flexible red leather, \$2.25, *net*. *Lea's Series of Pocket Text-Books.* Page 12.

**HAYEM (GEORGES) AND HARE (H. A.).** *PHYSICAL AND NATURAL THERAPEUTICS.* Heat, Electricity, Climate and Mineral Waters. Edited by H. A. HARE, M.D. Octavo, 414 pages, with 113 engravings. Cloth, \$3.

**HERMAN (G. ERNEST).** *FIRST LINES IN MIDWIFERY.* 12mo., 198 pages, with 80 engravings. Cloth, \$1.25. See *Students' Series of Manuals.* Page 15.



- HERRICK (JAMES B.).** *A HANDBOOK OF DIAGNOSIS.* In one handsome 12mo. volume of 429 pages, with 80 engravings and 2 colored plates. Cloth, \$2.50.
- HERTER (C. A.).** *LECTURES ON CHEMICAL PATHOLOGY.* In one 12 mo. volume of 454 pages. Cloth, \$1.75, *net*.
- HILL (BERKELEY).** *SYPHILIS AND LOCAL CONTAGIOUS DISORDERS.* In one 8vo. volume of 479 pages. Cloth, \$3.25.
- HILLIER (THOMAS).** *A HANDBOOK OF SKIN DISEASES.* Second edition. In one royal 12mo. volume of 353 pages, with two plates. Cloth, \$2.25.
- HIRST (BARTON C.) AND PIERSOL (GEORGE A.).** *HUMAN MONSTROSITIES.* Magnificent folio, containing 220 pages of text and illustrated with 123 engravings and 39 large photographic plates from nature. In four parts, price each, \$5.
- HOBLYN (RICHARD D.).** *A DICTIONARY OF THE TERMS USED IN MEDICINE AND THE COLLATERAL SCIENCES.* Thirteenth edition. In one 12mo. volume of 845 pages. Cloth, \$3.00, *net*.
- HOLMES (TIMOTHY).** *A TREATISE ON SURGERY.* Fifth edition. Octavo, 1008 pages, with 428 engravings. Cloth, \$6; leather, \$7.
- *A SYSTEM OF SURGERY.* Edited by JOHN H. PACKARD, M.D. In three 8vo. volumes, 3137 pages, 979 engravings, 13 plates. Per volume, Cloth, \$6.
- HUNTINGTON (GEORGE S.).** *ABDOMINAL ANATOMY.* Imperial quarto, 590 pages, including 300 full-page plates. De luxe edition, \$10.00, *net*.
- HYDE (JAMES NEVINS) AND MONTGOMERY (FRANK H.).** *A PRACTICAL TREATISE ON DISEASES OF THE SKIN.* Sixth edition, thoroughly revised. Octavo, 832 pages, with 107 engravings and 27 full-page plates, 9 of which are colored. Cloth, \$4.50, *net*; leather, \$5.50, *net*; half morocco, \$6.00, *net*.
- JACKSON (GEORGE THOMAS).** *THE READY-REFERENCE HANDBOOK OF DISEASES OF THE SKIN.* Fourth edition. 12mo. volume of 617 pages, with 82 engravings, and 3 colored plates. Cloth, \$2.75, *net*.
- JAMIESON (W. ALLAN).** *DISEASES OF THE SKIN.* Third edition. Octavo, 656 pages, with 1 engraving and 9 double-page chromo-lithographic plates. Cloth, \$6.
- JEWETT (CHARLES).** *ESSENTIALS OF OBSTETRICS.* Second edition. 12mo., 385 pages, with 80 engravings and 5 colored plates. Cloth, \$2.25, *net*.
- *THE PRACTICE OF OBSTETRICS.* By American Authors. Second edition. One octavo volume of 775 pages, with 445 engravings in black and colors, and 35 full-page colored plates. Cloth, \$5.00; leather, \$6.00; half morocco, \$6.50.
- JULER (HENRY).** *A HANDBOOK OF OPHTHALMIC SCIENCE AND PRACTICE.* Second edition. Octavo, 549 pages, with 201 engravings, 17 chromo-lithographic plates, test-types and Color-Blindness Test. Cloth, \$5.50; leather, \$6.50.
- KELLY (A. O. J.)** *A MANUAL OF THE PRACTICE OF MEDICINE.* Octavo, about 600 pages, illustrated. *Preparing.*
- KIRK (EDWARD C.).** *OPERATIVE DENTISTRY.* Second edition. See *American Text-books of Dentistry*, page 2.
- KING (A. F. A.).** *A MANUAL OF OBSTETRICS.* Eighth edition. In one 12mo. volume of 612 pages, with 264 illustrations. Cloth, \$2.50, *net*.
- KLEIN (E.).** *ELEMENTS OF HISTOLOGY.* Fifth edition. In one pocket-size 12mo. volume of 506 pages, with 296 engravings. Cloth, \$2.00, *net*. *Students' Series of Manuals.* Page 15.
- KOPLIK (HENRY).** *DISEASES OF INFANCY AND CHILDHOOD.* Octavo, 675 pages with 169 engravings and 32 plates in black and colors. Cloth, \$5.00; leather, \$6.00, *net*. *Just Ready.*
- KÜSTNER (OTTO) AND BEYEA (HENRY D.).** *A TEXT-BOOK OF GYNECOLOGY.* Octavo, 400 pages, with 260 engravings. *Preparing.*



**LANDIS (HENRY G.).** *THE MANAGEMENT OF LABOR.* In one handsome 12mo. volume of 329 pages, with 28 illustrations. Cloth, \$1.75.

**LA ROCHE (R.).** *YELLOW FEVER.* In two 8vo. volumes of 1468 pages. Cloth, \$7.

**LEA (HENRY C.).** *CHAPTERS FROM THE RELIGIOUS HISTORY OF SPAIN; CENSORSHIP OF THE PRESS; MYSTICS AND ILLUMINATI; THE ENDEMONIADAS; EL SANTO NINO DE LA GUARDIA; BRINDA DE BARDAXI.* In one 12mo. volume of 522 pages. Cloth, \$2.50.

— *A HISTORY OF AURICULAR CONFESSION AND INDULGENCES IN THE LATIN CHURCH.* In three octavo volumes of about 500 pages each. Per volume, cloth, \$3.

— *THE MORISCOS OF SPAIN: THEIR CONVERSION AND EXPULSION.* In one royal 12mo. volume of about 425 pages. Extra cloth, \$2.25, net.

— *STUDIES IN CHURCH HISTORY.* New edition. 12mo., 605 pages. Cloth, \$2.50.

— *SUPERSTITION AND FORCE; ESSAYS ON THE WAGER OF LAW, THE WAGER OF BATTLE, THE ORDEAL AND TORTURE.* Fourth edition, thoroughly revised. In one royal 12mo. volume of 629 pages. Cloth, \$2.75.

**LEA'S SERIES OF MEDICAL EPITOMES.** Covering the entire field of medicine and surgery in twenty convenient volumes of about 250 pages each, amply illustrated and written by prominent teachers and specialists. Compendious, authoritative and modern. Following each chapter is a series of questions which will be found convenient in quizzing. The Series is constituted as follows:

Hale's Anatomy. Guenther's Physiology. McGlannan's Chemistry and Physics. Kiepe's Materia Medica and Therapeutics. Dayton's Practice of Medicine. Hollis's Physical Diagnosis. Arneill's Clinical Diagnosis and Urinalysis. Nagle's Nervous and Mental Diseases. Wathen's Histology. Stenhouse's Pathology. Archinard's Bacteriology. Magee and Johnson's Surgery. Alling, Griffen and Ferguson's Eye, Ear, Nose and Throat. Schmidt's Genito-Urinary and Venereal Diseases. Schalek's Dermatology. Pedersen's Gynecology. Manton's Obstetrics. Tuley's Pediatrics. Dwight's Jurisprudence. Dwight's Toxicology.

**LEA'S SERIES OF POCKET TEXT-BOOKS.** See page 12.

**LE FEVRE (EGBERT).** *A TEXT-BOOK OF PHYSICAL DIAGNOSIS.* 12mo., 450 pages, 74 engravings, 12 plates. Cloth, \$2.25, net.

**LONG (ELI).** *DENTAL MATERIA MEDICA AND THERAPEUTICS.* 12mo., 321 pages, 6 engravings, 18 plates. Cloth, \$3.00, net.

**LOOMIS (ALFRED L.) AND THOMPSON (W. GILMAN),** Editors. *A SYSTEM OF PRACTICAL MEDICINE.* In Contributions by Various American Authors. In four very handsome octavo volumes of about 900 pages each, fully illustrated in black and colors. Per volume, cloth, \$5; leather, \$6; half Morocco, \$7. *For sale by subscription only.* Full prospectus free on application.

**LYMAN (HENRY M.).** *THE PRACTICE OF MEDICINE.* In one very handsome octavo volume of 925 pages with 170 engravings. Cloth, \$4.75; leather, \$5.75.

**MACKENZIE (JOHN NOLAND).** *THE DISEASES OF THE NOSE AND THROAT.* Octavo, of about 600 pages, richly illustrated. *Preparing.*

**MAISCH (JOHN M.).** *A MANUAL OF ORGANIC MATERIA MEDICA.* Seventh edition, thoroughly revised by H. C. C. MAISCH, Ph.G., Ph.D. In one 12mo. of 512 pages, with 285 engravings. Cloth, \$2.50, net.

**MALSBARY (GEO. E.).** *A POCKET TEXT-BOOK OF THEORY AND PRACTICE OF MEDICINE.* 12mo. 405 pages, with 45 illustrations. Cloth, \$1.75, net; flexible red leather, \$2.25, net. *Lea's Series of Pocket Text-Books.* Page 12.



- MANTON (W. P.).** *AN EPITOME OF OBSTETRICS.* 12mo., 265 pages, 82 illustrations. Cloth, \$1.00, net. *Lea's Series of Medical Epitomes.* See page 10.
- MANUALS.** See *Students' Quiz Series*, page 14; *Students' Manuals*, page 15; *Clinical Manuals*, page 13; *Medical Epitomes*, page 10; *Pocket Text-Books*, page 12.
- MARSH (HOWARD).** *DISEASES OF THE JOINTS.* In one 12mo. volume of 468 pages, with 64 engravings and a colored plate. Cloth, \$2. See *Series of Clinical Manuals*, page 13.
- MARTIN (EDWARD.)** *SURGICAL DIAGNOSIS.* One 12mo. volume of 400 pages, richly illustrated. *Preparing.*
- MARTIN (WALTON) AND ROCKWELL (W. H., JR.).** *A POCKET TEXT-BOOK OF CHEMISTRY AND PHYSICS.* 12mo. 366 pages, with 137 illustrations. Cloth, \$1.50, net; flexible leather, \$2.00, net. *Lea's Series of Pocket Text-Books.* Page 12.<sup>3</sup>
- MCGLANNAN (A.).** *AN EPITOME OF CHEMISTRY AND PHYSICS.* 12mo, 250 pages, illustrated. Cloth, \$1.00, net. *Lea's Series of Medical Epitomes.* See page 10.
- MEDICAL NEWS POCKET FORMULARY.** See page 1. \$1.50, net.
- MITCHELL (JOHN K.).** *REMOTE CONSEQUENCES OF INJURIES OF NERVES AND THEIR TREATMENT.* 12mo., 239 pages, 12 illustrations. Cloth, \$1.75.
- MITCHELL (S. WEIR).** *CLINICAL LESSONS ON NERVOUS DISEASES.* 12mo., 299 pages, with 17 engravings and 2 colored plates. Cloth, \$2.50.
- MORRIS (MALCOLM).** *DISEASES OF THE SKIN.* Second edition. 12mo., 601 pages, with 10 chromo-lithographic plates and 26 engravings. Cloth, \$3.25, net.
- MORROW (PRINCE A.).** *THE RELATIONS OF VENEREAL DISEASES -WITH MARRIAGE.* Octavo, about 450 pages, with many illustrations. *Preparing.*
- MULLER (J.).** *PRINCIPLES OF PHYSICS AND METEOROLOGY.* In one large 8vo. volume of 623 pages, with 538 engravings. Cloth, \$4.50.
- MUSSER (JOHN H.).** *A TREATISE ON MEDICAL DIAGNOSIS,* for Students and Physicians. Fourth edition. Octavo, 1104 pages, with 250 engravings and 49 full-page colored plates. Cloth, \$6.00; leather, \$7.00; half morocco, \$7.50, net.
- NATIONAL DISPENSATORY.** See *Stillé, Maisch & Caspari*, page 14.
- NATIONAL FORMULARY.** See *National Dispensatory*, page 14.
- NATIONAL MEDICAL DICTIONARY.** See *Billings*, page 3.
- NETTLESHIP (E.).** *DISEASES OF THE EYE.* Sixth American from sixth English edition. Thoroughly revised. 12mo., 562 pages, with 192 engravings, 5 colored plates, test-types, formulæ and color-blindness test. Cloth, \$2.25, net.
- NICHOLS (JOHN B.) AND VALE (F. P.).** *A POCKET TEXT-BOOK OF HISTOLOGY AND PATHOLOGY.* 12mo. of 459 pages, with 213 illustrations. Cloth, \$1.75, net; flexible leather, \$2.25, net. *Lea's Series of Pocket Text-Books.* Page 12.
- NORRIS (WM. F.) AND OLIVER (CHAS. A.).** *TEXT-BOOK OF OPHTHALMOLOGY.* In one octavo volume of 641 pages, with 357 engravings and 5 colored plates. Cloth, \$5; leather, \$6.
- OWEN (EDMUND).** *SURGICAL DISEASES OF CHILDREN.* In one 12mo. volume of 525 pages, with 85 engravings and 4 colored plates. Cloth, \$2. See *Series of Clinical Manuals*, page 13.
- PARK (WILLIAM H.).** *BACTERIOLOGY IN MEDICINE AND SURGERY.* 12mo., 688 pages, 87 engravings in black and colors, 2 colored plates. Cloth, \$3.00, net.
- PARK (ROSWELL), Editor.** *A TREATISE ON SURGERY,* by American Authors. For Students and Practitioners of Surgery and Medicine. Third edition. In one large octavo volume of 1408 pages, with 692 engravings and 64 plates. Cloth, \$7.00; leather, \$8.00, net. Published also in 2 volumes at \$3.75, net, per volume, cloth.

**PARVIN (THEOPHILUS).** *THE SCIENCE AND ART OF OBSTETRICS.* Third edition. In one handsome octavo volume of 677 pages, with 267 engravings and 2 colored plates. Cloth, \$4.25; leather, \$5.25.

**PEPPER'S SYSTEM OF MEDICINE.** See page 2.

**PEPPER (A. J.).** *SURGICAL PATHOLOGY.* In one 12mo volume of 511 pages, with 81 engravings. Cloth, \$2. See *Students' Series of Manuals*, page 15.

**PICK (T. PICKERING).** *FRACTURES AND DISLOCATIONS.* In one 12mo volume of 530 pages, with 93 engravings. Cloth, \$2. See *Series of Clinical Manuals*, p. 13.

**PLAYFAIR (W. S.).** *THE SCIENCE AND PRACTICE OF MIDWIFERY.* Seventh American from the Ninth English edition. Octavo, 700 pages, with 207 engravings and 7 full page plates. Cloth, \$3.75; leather, \$4.75, *net*.

**POLITZER (ADAM).** *A TEXT-BOOK OF THE DISEASES OF THE EAR AND ADJACENT ORGANS.* Third American from the Fourth German edition. In one octavo volume of 896 pages, with 346 engravings. Cloth, \$7.50, *net*. *Just Ready.*

**POCKET FORMULARY.** Fifth edition. See page 1.

**POCKET TEXT-BOOK SERIES** covers the entire domain of medicine in eighteen volumes of 350 to 525 pages each, written by teachers in leading American medical colleges. Issued under the editorial supervision of BERN B. GALLAUDET, M.D., of the College of Physicians and Surgeons, New York. Thoroughly modern and authoritative, concise and clear, amply illustrated with engravings and plates, handsomely printed and bound. The series is constituted as follows: Anatomy; Physiology; Chemistry and Physics; Histology and Pathology; Materia Medica, Therapeutics, Medical Pharmacy, Prescription Writing and Medical Latin; Practice; Diagnosis; Nervous and Mental Diseases; Surgery; Venereal Diseases; Skin Diseases; Eye, Ear, Nose and Throat; Obstetrics; Gynecology; Diseases of Children; Bacteriology; Nursing; Massage. Special circular free on application.

**POTTS (CHAS. S.).** *A POCKET TEXT-BOOK OF NERVOUS AND MENTAL DISEASES.* 12mo. of 455 pages, with 88 illustrations. Cloth, \$1.75, *net*; flexible leather, \$2.25, *net*. Lea's Series of Pocket Text-Books, page 12.

— *A TEXT-BOOK ON MEDICINE AND SURGICAL ELECTRICITY.* Octavo, about 350 pages, amply illustrated. *Shortly.*

**POSEY (W. C.) AND WRIGHT (JONATHAN).** *A TREATISE ON THE EYE, NOSE, THROAT AND EAR.* Octavo, 1251 pages, richly illustrated with 650 engravings and 35 plates in black and colors. Cloth, \$7.00; leather, \$8.00, *net*. *Just Ready.* Published also in two volumes. Vol. I., Posey on the Eye. Cloth, \$4.00, *net*. Vol. II., Wright on the Nose, Throat and Ear. Cloth, \$3.50, *net*.

**PROGRESSIVE MEDICINE.** See page 1. Per annum, \$10.00.

**PURDY (CHARLES W.).** *BRIGHT'S DISEASE AND ALLIED AFFECTIONS OF THE KIDNEY.* In one octavo volume of 288 pages, with 18 engravings. Cloth, \$2.

**PYE-SMITH (PHILIP H.).** *DISEASES OF THE SKIN.* In one 12mo. volume of 407 pages, with 28 illustrations, 18 of which are colored. Cloth, \$2.

**QUIZ SERIES.** See *Students' Quiz Series*, page 14.

**RALFE (CHARLES H.).** *CLINICAL CHEMISTRY.* In one 12mo. volume of 314 pages, with 16 engravings. Cloth, \$1.50. See *Students' Series of Manuals*, page 14.

**REMSEN (IRA).** *THE PRINCIPLES OF THEORETICAL CHEMISTRY.* Fifth edition thoroughly revised. In one 12mo. volume of 326 pages. Cloth, \$2.

**REYNOLDS (EDWARD) AND NEWELL (F. S.).** *MANUAL OF PRACTICAL OBSTETRICS.* Octavo, 531 pages, 253 engravings and 3 plates. Cloth, \$3.75, *net*. *Just Ready.*

Philadelphia, 706, 708 and 710 Sansom St.,—New York, 111 Fifth Avenue.



**RICHARDSON (MAURICE H.).** *A PRACTICAL TREATISE ON ABDOMINAL SURGERY.* Octavo, about 800 pages, profusely illustrated with engravings and colored plates. *Preparing.*

**RICHARDSON (BENJAMIN WARD).** *PREVENTIVE MEDICINE.* In one octavo volume of 729 pages. Cloth, \$4.

**ROBERTS (JOHN B.).** *THE PRINCIPLES AND PRACTICE OF MODERN SURGERY.* Second edition. In one octavo volume of 838 pages, with 474 engravings and 8 plates. Cloth, \$4.25, *net*; leather, \$5.25, *net*.

**ROCKWELL (W. H., Jr.).** *A POCKET TEXT-BOOK OF ANATOMY.* 12mo., 600 pages, illustrated. Cloth, \$2.25; flexible leather, \$2.75, *net.* *Lea's Series of Pocket Text-Books.* Page 12.

**ROSS (JAMES).** *THE DISEASES OF THE NERVOUS SYSTEM.* Octavo, 726 pages, with 184 engravings. Cloth, \$4.50; leather, \$5.50.

**SCHAFER (EDWARD A.).** *THE ESSENTIALS OF HISTOLOGY, DESCRIPTIVE AND PRACTICAL.* Sixth edition. Octavo, 426 pages, with 463 illustrations. Cloth, \$3, *net.*

— *A COURSE OF PRACTICAL HISTOLOGY.* Second edition. In one 12mo. volume of 307 pages, with 59 engravings. Cloth, \$2.25.

**SCHALEK (ALFRED).** *AN EPITOME OF DERMATOLOGY.* 12mo., 225 pages, 34 illustrations. Cloth, \$1.00, *net.* *Lea's Series of Medical Epitomes.* See page 10.

**SCHLEIF (WM.).** *A POCKET TEXT-BOOK OF MATERIA MEDICA, THERAPEUTICS, PRESCRIPTION WRITING, MEDICAL LATIN AND MEDICAL PHARMACY.* Second edition. 12mo., 380 pages. Cloth, \$1.75; flexible leather, \$2.25, *net.* *Lea's Series of Pocket Text-Books.* Page 12.

**SCHMAUS (HANS.) AND EWING (JAMES).** *PATHOLOGY AND PATHOLOGICAL ANATOMY.* Sixth edition. Octavo, 602 pages, with 351 illustrations, including 34 colored inset plates. Cloth, \$4.00, *net.* *Just Ready.*

**SCHMIDT (LOUIS E.).** *AN EPITOME OF GENITO-URINARY AND VENEREAL DISEASES.* 12mo., 249 pages, 21 illustrations. Cloth, \$1.00, *net.* *Lea's Series of Medical Epitomes.* See page 10.

**SCHREIBER (JOSEPH).** *A MANUAL OF TREATMENT BY MASSAGE AND METHODICAL MUSCLE EXERCISE.* Translated by WALTER MENDELSON, M.D., of New York. Octavo, 274 pages, with 117 engravings.

**SCOTT (R. J. E.).** See State Board Examinations, page 14.

**SENN (NICHOLAS).** *SURGICAL BACTERIOLOGY.* Second edition. In one octavo volume of 268 pages, with 13 plates, 10 of which are colored, and 9 engravings. Cloth, \$2.

**SERIES OF CLINICAL MANUALS.** A Series of Authoritative Monographs on Important Clinical Subjects, in 12mo. volumes of about 550 pages, well illustrated. The following volumes are now ready: CARTER and FROST's Ophthalmic Surgery, \$2.25; MARSH on Diseases of the Joints, \$2; OWEN on Surgical Diseases of Children, \$2; PICK on Fractures and Dislocations, \$2.

For separate notices, see under various authors' names.

**SERIES OF POCKET TEXT-BOOKS.** See page 12.

**SERIES OF STATE BOARD EXAMINATIONS.** See page 14.

**SERIES OF STUDENTS' MANUALS.** See page 15.

**SIMON (CHARLES E.).** *CLINICAL DIAGNOSIS, BY MICROSCOPICAL AND CHEMICAL METHODS.* Fourth revised edition. Octavo, 608 pages, with 139 engravings and 19 full-page plates in colors. Cloth, \$3.75, *net.*

— *PHYSIOLOGICAL CHEMISTRY.* In one octavo volume of 453 pages. Cloth, \$3.25, *net.*

Philadelphia, 706, 708 and 710 Sansom St.—New York, 111 Fifth Avenue.

**SIMON (W.).** *MANUAL OF CHEMISTRY.* A Guide to Lectures and Laboratory Work. A Text-book specially adapted for Students of Medicine and Pharmacy. Seventh edition. In one 8vo. volume of 613 pages, with 64 engravings and 8 plates showing colors of 64 tests and a spectra plate. Cloth, \$3.00, *net*.

**SLADE (D. D.).** *DIPHTHERIA; ITS NATURE AND TREATMENT.* Second edition. In one royal 12mo. volume, 158 pages. Cloth, \$1.25.

**SMITH (J. LEWIS).** *THE DISEASES OF INFANCY AND CHILDHOOD.* Eighth edition, thoroughly revised and rewritten and greatly enlarged. 8vo., 983 pages, with 273 illustrations and 4 full-page plates. Cloth, \$4.50; leather, \$5.50.

**SMITH (STEPHEN).** *OPERATIVE SURGERY.* Second and thoroughly revised edition. In one octavo vol. of 892 pages, with 1005 engravings. Cloth, \$4; leather, \$5.

**SOLLY (S. EDWIN).** *A HANDBOOK OF MEDICAL CLIMATOLOGY.* Octavo, 462 pages, with engravings and 11 full-page plates. Cloth, \$4.00.

**STARR (M. ALLEN).** *A TREATISE ON ORGANIC NERVOUS DISEASES.* Octavo, about 700 pages, with 300 engravings and 27 colored plates. *Preparing.*

**STATE BOARD EXAMINATION SERIES.** Classified and Edited by R. J. E. SCOTT, A.M., M.D. A series of handy, well arranged, 12mo. volumes of about 200 pages each, bound in flexible cloth and printed on paper suitable for either pen or pencil, every other page being left blank for memoranda. The respective volumes cover the subjects of Anatomy; Physiology and Hygiene; Chemistry; Surgery; Obstetrics; Pathology and Diagnosis; Practice, Materia Medica and Therapeutics. *Shortly.*

**STILLE (ALFRED).** *CHOLERA; ITS ORIGIN, HISTORY, CAUSATION, SYMPTOMS, LESIONS, PREVENTION AND TREATMENT.* In one 12mo. volume of 163 pages, with a chart showing routes of previous epidemics. Cloth, \$1.25.

— *THERAPEUTICS AND MATERIA MEDICA.* Fourth and revised edition. In two octavo volumes, containing 1936 pages. Cloth, \$10.

**STILLE (ALFRED), MAISCH (JOHN M.) AND CASPARI (CHAS. JR.).** *THE NATIONAL DISPENSATORY:* Containing the Natural History, Chemistry, Pharmacy, Actions and Uses of Medicines, including those recognized in the latest Pharmacopœias of the United States, Great Britain and Germany, with numerous references to the French Codex. Fifth edition, revised and enlarged in accordance with and embracing the new U. S. Pharmacopœia, Seventh Decennial Revision. With Supplement containing the new edition of the *National Formulary.* Imperial octavo, 2025 pages, with 320 engravings. Cloth, \$7.25; leather, \$8. With Thumb Index. Cloth, \$7.75; leather, \$8.50.

**STIMSON (LEWIS A.).** *A MANUAL OF OPERATIVE SURGERY.* Fourth edition. In one royal 12mo. volume of 581 pages, with 293 engravings. Cloth, \$3.00, *net*.

— *A TREATISE ON FRACTURES AND DISLOCATIONS.* Third edition. In one handsome octavo volume of 842 pages, with 336 engravings and 32 full-page plates. Cloth, \$5 *net*; leather, \$6, *net*; half morocco, \$6.50, *net*.

**STUDENTS' QUIZ SERIES.** A Series of Manuals in question and answer form, for Students and Practitioners, covering the essentials of medical science. Thirteen volumes, pocket size, convenient, authoritative, well illustrated, handsomely bound in cloth, and issued at a low price. 1. Anatomy (double number); 2. Physiology; 3. Chemistry and Physics; 4. Histology, Pathology and Bacteriology; 5. Materia Medica and Therapeutics; 6. Practice of Medicine; 7. Surgery (double number); 8. Genito-Urinary and Venereal Diseases; 9. Diseases of the Skin; 10. Diseases of the Eye, Ear, Throat and Nose; 11. Obstetrics; 12. Gynecology; 13. Diseases of Children. Price, \$1 each, except Nos. 1 and 7, *Anatomy and Surgery*, which being double numbers are priced at \$1.75 each. Full specimen circular on application to publishers.



**STUDENTS' SERIES OF MANUALS.** A Series of Manuals by Eminent Teachers or Examiners. The volumes are pocket-size 12mos. of from 300-540 pages, profusely illustrated, and bound in red limp cloth. The following volumes may now be announced: HERMAN's First Lines in Midwifery, \$1.25; BRUCE's Materia Medica and Therapeutics (sixth edition), \$1.50, *net*; KLEIN's Elements of Histology (5th edition), \$2.00, *net*; PEPPER's Surgical Pathology, \$2; TREVES' Surgical Applied Anatomy, \$2.00; RALFE's Clinical Chemistry, \$1.50.

For separate notices, see under various authors' names.

**STURGES (OCTAVIUS).** *AN INTRODUCTION TO THE STUDY OF CLINICAL MEDICINE.* In one 12mo. volume. Cloth, \$1.25.

**SUTER (W. NORWOOD).** *A MANUAL OF REFRACTION AND MOTILITY.* 12mo., 382 pages, 101 engravings, 4 colored plates. Cloth, \$2.00, *net*. *Just Ready.*

**SUTTON (JOHN BLAND).** *SURGICAL DISEASES OF THE OVARIES AND FALLOPIAN TUBES.* Including Abdominal Pregnancy. In one 12mo. volume of 513 pages, with 119 engravings and 5 colored plates. Cloth, \$3.

**SZYMONOWICZ (L.) AND MacCALLUM (J. BRUCE).** *A TEXT-BOOK OF HISTOLOGY OF THE HUMAN BODY:* including Microscopical Technique. Octavo, 437 pages, with 277 original engravings and 57 inset plates in black and colors, containing 81 figures. Cloth, \$4.75, *net*. *Just Ready.*

**TAIT (LAWSON).** *DISEASES OF WOMEN AND ABDOMINAL SURGERY.* Vol. I. contains 554 pages, 62 engravings, and 3 plates. Cloth, \$3.

**TAYLOR (ALFRED S.).** *MEDICAL JURISPRUDENCE.* From the twelfth English edition, revised by CLARK BELL, Esq., of the N. Y. Bar. Octavo, 831 pages, with 54 engravings and 8 full-page plates. Cloth, \$4.50; leather, \$5.50.

**TAYLOR (ROBERT W.).** *GENITO-URINARY AND VENEREAL DISEASES AND SYPHILIS.* Second edition. Octavo, 720 pages, with 135 engravings and 27 colored plates. Cloth, \$5.00; leather, \$6.00; half morocco, \$6.50, *net*.

— *A PRACTICAL TREATISE ON SEXUAL DISORDERS IN THE MALE AND FEMALE.* Second edition. Octavo, 434 pages, with 91 engravings and 13 plates. Cloth, \$3.00, *net*.

— *A CLINICAL ATLAS OF VENEREAL AND SKIN DISEASES.* Including Diagnosis, Prognosis and Treatment. In eight large folio parts, measuring 14 x 18 inches, and comprising 213 beautiful figures on 58 full-page chromo-lithographic plates, 85 fine engravings, and 425 pages of text. Bound in one volume, half Turkey Morocco, \$28. Specimen plates by mail.

**TAYLOR (SEYMOUR).** *INDEX OF MEDICINE.* A Manual for the use of Senior Students and others. In one large 12mo. volume of 802 pages. Cloth, \$3.75.

**THOMAS (T. GAILLARD) AND MUNDE (PAUL F.).** *A PRACTICAL TREATISE ON THE DISEASES OF WOMEN.* Sixth edition, thoroughly revised. Octavo 824, pages, with 347 engravings. Cloth, \$5; leather, \$6.

**THOMPSON (W. GILMAN).** *A TEXT-BOOK OF PRACTICAL MEDICINE.* For Students and Practitioners. Second edition. Octavo, 1014 pages, with 59 illustrations. Cloth, \$5.00, leather, \$6.00, half morocco, \$6.50, *net*. *Just Ready.*

**THOMPSON (SIR HENRY).** *THE PATHOLOGY AND TREATMENT OF STRICTURE OF THE URETHRA AND URINARY FISTULÆ.* From the third English edition. Octavo, 359 pages, with 47 engravings and 3 plates. Cloth, \$3.50.

**TIRARD (NESTOR).** *MEDICAL TREATMENT OF DISEASES AND SYMPTOMS.* Handsome octavo volume of 627 pages. Cloth, \$4.00, *net*.

**TREVES (FREDERICK).** *OPERATIVE SURGERY.* In two 8vo. volumes containing 1550 pages, with 422 illustrations. Cloth, \$9; leather, \$11.

**TREVES (FREDERICK).** *A SYSTEM OF SURGERY.* In Contributions by Twenty-five English Surgeons. In two large octavo volumes, containing 2298 pages, with 960 engravings and 4 full-page plates. Per set, cloth, \$16.

— *SURGICAL APPLIED ANATOMY.* New edition. 12mo., 577 pages, 80 engravings. Cloth, \$2.00, net. See *Students' Series of Manuals*, page 15.

**TUTTLE (GEO. M.).** *A POCKET TEXT-BOOK OF DISEASES OF CHILDREN.* 12mo. 374 pages, with 5 plates. Cloth, \$1.50, net; flexible leather, \$2.00, net. *Lea's Series of Pocket Text-Books.* Page 12.

**VAUGHAN (VICTOR C.) AND NOVY (FREDERICK G.).** *CELLULAR TOXINS, or the Chemical Factors in the Causation of Disease.* New (4th) edition. 12mo., 480 pages with 6 engravings. Cloth, \$3, net.

**VEASEY (CLARENCE A.).** *A MANUAL OF OPHTHALMOLOGY.* 12mo., 410 pages, 194 engravings, 10 colored plates. Cloth, \$2.00, net. *Just Ready.*

**VISITING LIST. THE MEDICAL NEWS VISITING LIST for 1903.** Four styles: Weekly (dated for 30 patients); Monthly (undated for 120 patients per month); Perpetual (undated for 30 patients each week); and Perpetual (undated for 60 patients each week). The 60-patient book consists of 256 pages of assorted blanks. The first three styles contain 32 pages of important data, thoroughly revised, and 160 pages of assorted blanks. Each in one volume, price, \$1.25. With thumb-letter index for quick use, 25 cents extra. For special combination rates see page 1.

**WATSON (THOMAS).** *LECTURES ON THE PRINCIPLES AND PRACTICE OF PHYSIC.* Fifth edition with additions by H. HARTSHORNE, M.D. In two 8vo. volumes of 1840 pages, with 190 engravings. Cloth, \$9.

**WHARTON (HENRY R.).** *MINOR SURGERY AND BANDAGING.* Fifth edition. 12mo., 640 pages, with 509 engravings, many of which are photographic. Cloth, \$3.00, net.

**WHITMAN (ROYAL).** *ORTHOPEDIC SURGERY.* One octavo volume of 642 pages, with 447 illustrations, mostly original. Cloth, \$5.50, net.

**WHITLA (WILLIAM).** *DICTIONARY OF TREATMENT.* Octavo of 917 pages. Cloth, \$4.

**WILLIAMS (DAWSON).** *MEDICAL DISEASES OF INFANCY AND CHILDHOOD.* Second edition specially revised for America by F. S. Churchill, A.M., M.D. Octavo, 533 pages, 52 engravings and 2 colored plates. Cloth, \$3.50, net.

**WILSON (ERASMUS).** *A SYSTEM OF HUMAN ANATOMY.* Revised edition, octavo, 616 pages, with 397 engravings. Cloth, \$4; leather, \$5.

**WINCKEL ON PATHOLOGY AND TREATMENT OF CHILDREN.** In one octavo volume of 484 pages. Cloth, \$4.

**WOOLSEY (GEORGE).** *APPLIED SURGICAL ANATOMY REGIONALLY PRESENTED.* Octavo, 511 pages, with 125 original illustrations in black and colors. Cloth, \$5.00; leather, \$6.00, net. *Just Ready.*

**YEAR-BOOK OF TREATMENT FOR 1898.** In contributions by 24 well-known medical writers. 12mo., 488 pages. Cloth, \$1.50.

**YOUNG (JAMES K.).** *ORTHOPEDIC SURGERY.* In one 8vo volume of 475 pages, with 286 illustrations. Cloth, \$4; leather, \$5.

**ZAPFFE (FRED. C.)** *A POCKET TEXT-BOOK OF BACTERIOLOGY.* 12mo., 350 pages with 146 engravings and 7 colored plates. Cloth, \$1.50; flexible leather, \$2.00, net. *Just Ready.* *Lea's Series of Pocket Text-Books.* Page 12.









25M-2-52-86267

